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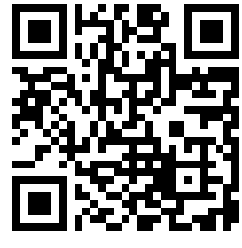

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THE

CLINICAL JOURNAL

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DEBATE

ON

DIABETIC AND NON-DIABETIC GLYCOSURIA.

Held under the auspices of the Chelsea Clinical Society at the Jenner Institute of Preventive Medicine, March 10th and 17th, 1903.

Dr. C. C. Gibbes, President, in the Chair.

DR. HALE WHITE: When, sir, I accepted the honour of an invitation to speak at this debate, I never dreamed it would fall to my lot to open it. Had I done so, I should not have dared to accept, for the subject is so vast and the difficulties are so great. As the President has told us that the whole subject of diabetes is under discussion, and as I do not know to what points subsequent speakers will allude, perhaps I had better lay before you points connected with the whole subject of diabetes.

The first thing that naturally attracts our attention is the recognition of dextrose in the urine. The universally employed copper-reduction test is unsatisfactory. As an example I may mention that about twenty years ago, when the nervous theory for diabetes was in the ascendant, it was urged in support of it that the urine of lunatics frequently contained sugar. Dr. G. H. Savage and I* showed this was not so; the mistake probably arose from confounding the reduction given by kreatin and the other reducing bodies in the urine with that given by sugar. We all know the difficulty in life-insurance work of estimating the value of a dirty, greenish-brown precipitate given by boiling urine with copper solution and an alkali. We have probably all come across people condemned as diabetic who are not, and the mistake has arisen either from the use of a stale copper solution, or because of an abundance of reducing bodies in the urine which are not sugar. Those

* 'Path. Trans.,' vol. xxxiv, p. 348.

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who are alcaptonuric are usually condemned as diabetic. The polarimeter, fermentation, and the formation of osazone crystals are all excellent tests; but the first requires an expert; the second is tedious; the third needs time—there may be delay in the formation of the crystals,—and glycuronic acid also gives crystals, which, however, are in rosettes of thick, plump needles, not bundles of long needles like those of phenyl-glucosazone. We want some one to get up and tell us he has found a test by which small quantities of dextrose can be detected in the urine as easily as small amounts of albumen. We also want to know more about the appearance of glycuronic acid* in the urine. Do some people pass it, and later on become glycosuric? One case I have seen suggests, but does not prove, that this may be so. A woman whom I saw ten years ago complained of thirst and dry skin; the urine had a low specific gravity (1008), contained no albumen, but gave on many occasions an abundant brick-red precipitate on boiling with Fehling's solution. It did not ferment. I saw her again a few weeks ago, and she is now passing genuine sugar, but the urine does not give a ferric-chloride reaction. Bearing on this we have Futcher's paper,† in which he shows that both fusel oil and tertiary amyl alcohol cause both dextrose and glycuronic acid to appear in the urine. Then, too, it appears that some sufferers from diabetes pass a little glycuronic acid; and it has been even stated that minute amounts of this acid may be found in healthy urine. Indeed, it must be remembered that there is every reason to believe that its appearance depends upon its being provided with something to combine with, rather than on any disturbance of, carbohydrate metabolism. Have any here present practical experience of Bial's‡ test for it? There is, I think, no doubt that in most cases the presence of glycuronic acid is asserted on insufficient grounds. A copper-reducing body is found which is not sugar, and it is assumed to be glycuronic acid.

Allied to this is another subject on which we want further information, namely, that we occasionally see patients suffering from exophthalmic goitre who for a long while pass an abundance of sugar in the urine. For example, I saw a patient ten years ago with marked exophthalmic goitre and glycosuria. Her doctor writes now to say the glycosuria persisted two years, slowly the symptoms of exophthalmic goitre have disappeared, and now she is "practically well." Some authors have maintained that glycosuria is particularly easily induced in patients with exophthalmic goitre, but von Schrötter* gave such a patient grape-sugar in doses of 200 grammes without any appearing in the urine. I have notes of one patient with exophthalmic goitre whose father had diabetes, and of another who has two sisters who have diabetes. Probably the fact that polyuria, thirst, and great hunger are frequent symptoms met with in exophthalmic goitre is unconnected with the glycosuria, for they occur independently of it; and in support of this is the fact that French,† making observations on a patient of mine with severe traumatic diabetes insipidus, was unable to produce glycosuria even by giving large amounts of sugar.

We want information about the occurrence of other sugars than dextrose in the urine. Clinically three appear important—lævulose, lactose, and pentose. Lævulose I will refer to presently. Can any speakers give us definite information about the after-history of women who during suckling pass lactose in their urine? It is stated that of those who develop glycosuria during pregnancy, recovery takes place in three quarters with a tendency for the glycosuria to recur in other pregnancies: but we want to know of the quarter who do not recover; do they become genuine diabetics or continue to pass lactose? Also we want to know if a quarter is not too high a proportion of those that do not recover; probably it is. Very few, if any, observations have been made in England on pentosuria. Pentose appears to exist in the urine in two condi-

* See a case by Ashdown, 'Brit. Med. Journ.,' vol. i, 1890, p. 167; also Bial, "Ueber Bildung und Ausscheidung der Glycuronicsäure," 'Zeit. f. klin. Med.,' Bd. xlvii, Hfte. 5 and 6.

† Futcher, "Fusel-oil Poisoning, with Special Reference to the Copper-reducing Substances eliminated in the Urine," 'American Medicine,' August 10th, 1901.

‡ Bial's reagent consists of orcin, concentrated hydrochloric acid, and ferric chloride. Take equal quantities of the reagent and of urine. On just raising to boiling, pentose gives a green-blue colour readily taken up by amyl alcohol, and with a characteristic spectrum. If boiling is continued, glycuronic acid gives the same reaction.

* "Zum Symptomencomplex des Morbus Basedowii," 'Zeitschrift für klinische Medicin,' Bd. xlviii, Hft. 1.

† 'Guy's Hospital Reports,' vol. lvii, p. 133.

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tions, namely, in association with other sugars in diabetes—as, for example, d'Amato, who has recently recorded the case of a patient with a pancreatic calculus and diabetes, and in whom the urine contained both dextrose and pentose,—and in very rare cases as an independent condition in which, although pentose appears in the urine, the patient can utilise pentose. Brat* has recently recorded a case of pentosuria in a woman æt. 62, whose brother also had pentosuria. Some of the patients with pentosuria were morphomaniacs. Is pentosuria found in any large proportion of morphia takers? In passing I should like to suggest that as accuracy of language leads to accuracy of thought, we should, when we know the sugar in the urine to be dextrose, speak of dextrosuria, thus harmonising with lævulosuria, pentosuria, and lactosuria.

Talking of nomenclature leads me to ask for a more accurate use of the word diabetes. Even the use of the additional word mellitus does not help, for many speak of alimentary glycosuria as diabetes mellitus. As it is now, if I hear a patient has diabetes I do not know whether he has the comparatively harmless alimentary glycosuria or the dangerous disease diabetes in which he is liable to coma and passes oxybutyric acid and its derivatives in his urine. Fagge was of opinion that the word diabetes should be used only for the severer disease; and this would be best. However, I doubt whether now this is possible, but at any rate we might adopt Dr. Pavy's excellent suggestion and speak of alimentary diabetes, and use the phrase "composite diabetes" to indicate the cases which pass sugar and oxybutyric acid and its derivatives, of which aceto-acetic acid is recognised by the ferric-chloride reaction and acetone by the nitro-prusside of sodium reaction; but the detection of acetone is not nearly so important as the detection of aceto-acetic acid, for acetone is often present in persons who have no diabetes. It cannot be too strongly urged that no case of sugar in the urine has been even superficially examined unless the ferric-chloride test has been employed.

We badly want a series of cases recorded with a view of finding out what proportion of these alimentary cases become composite cases. Some will certainly appear to do so, for many an apparent alimentary case is really the early stage of a composite case; indeed, Ebstein believes that this is so of all alimentary cases. Dr. Pavy, too, is strongly of opinion that alimentary cases often, at least later, become composite unless properly treated. Properly treated, however, a patient suffering from the alimentary form may remain perfectly well for many years. I recently saw a man æt. 62 whose urine had a specific gravity of 1036; it contained much sugar, gave no ferric-chloride reaction, and he appeared perfectly well. He said that sugar was detected in 1885, and that he had dieted carefully for the last ten years. Such a case as this casts doubt on the view that hyperglycæmia is harmful. It is true that many a patient with this form of glycosuria has a little albuminuria and rigid arteries, that he is liable to gangrene or boils, and that he bears operations and illnesses badly; but are not all these, like the glycosuria, to be regarded as evidence of structural and functional decay rather than as due to the glycosuria? However, it must be remembered that the passage of sugar, being a loss of energy, is in itself harmful; but as the amount of sugar is usually small, this loss of energy is generally unimportant. It is strongly in support of the above view that this form of glycosuria is met with chiefly in elderly people. There is sometimes a tendency to treat these alimentary cases too severely. It is not necessary to reduce the carbohydrates in the food beyond the point that leads to their disappearance from the urine. But the urine should be more carefully examined than is usually the case, for an alimentary lævulosuria* has been described. A complete record of such a case, giving the effects of the administration of dextrose and lævulose, would be very valuable, and we should like information as to the value of Seliwanoff's test for lævulose.† To be strictly accurate, we ought to speak of alimentary dextrosuria and alimentary lævulosuria, as the case

* H. Brat, "Beitrag zur Kenntniss der Pentosurie und der Pentosenreaction," 'Zeitschrift für klinische Medicin,' Bd. xlvii, Hfte. 5 and 6.

* Rosin and Laband, "Ueber spontane Lävulosurie und Lävulosämie," 'Zeitschrift f. klin. med.,' Bd. xlvii, Hfte. 1 and 2.

† Seliwanoff's test is fully described in Rosin and Laband's paper. It consists in a bright red colour given after heating an equal part of urine with fuming hydrochloric acid and a little piece of resorcin. This reaction is only given by sugars allied to ketones, and therefore by lævulose. Acetone, although a ketone, does not give it. There is no other body than lævulose that ferments and gives this reaction.

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may be. It will be noticed I have assumed (what I believe to be true) that while many patients with so-called alimentary glycosuria are really in the early stages of composite diabetes, yet others are examples of a much less harmful condition, which always remains alimentary glycosuria, never passes on to composite diabetes, and lacks the serious metabolic phenomena of this disease. Whether this is so is a very fair subject for discussion.

The occurrence of sugar in the urine due to diseases or injuries of the nervous system is of little clinical interest. It is usually slight and transitory. If the nervous lesion causes coma, and sugar is found in the urine, the case may at first be mistaken for diabetic coma. I was called to a case not long ago in which this mistake had been made, but the failure to find either acetone or aceto-acetic acid in the urine showed the case to be one of nervous glycosuria. The patient died from cerebral hæmorrhage, and it was found that blood had passed down the iter into the fourth ventricle. Dr. Beddard has kindly directed my attention to a similar case,* in which acetone chanced to be in the urine; aceto-acetic acid was not looked for, but doubtless ferric chloride would have shown its absence. Testing for the ferric-chloride reaction will always prevent a mistake between a nervous lesion causing coma with sugar in the urine and diabetic coma.

We now pass on to consider diabetes proper, or, as Dr. Pavy calls it, "composite diabetes." This may be defined as a disease in which dextrose is present in the blood and urine, and in which, if the disease is untreated, ultimately lævo-rotatory β-hydroxybutyric acid appears in the blood and urine, and the output of carbohydrates in the urine is greater than can be accounted for by those in the food. By far the most important sugar in the urine is dextrose, but small quantities of lævulose, pentose, combined glycuronic acid, and other closely allied bodies are sometimes present. We know there is a great excess of sugar in the blood, and that this excess is very largely due, and probably in many cases (at any rate in the early stages) entirely due, to the failure of the body to utilise the sugar; but even in many severe cases the power to utilise sugar is not entirely lost. But this is not in all cases the entire explanation of the presence of sugar in the blood, for in a severe case of diabetes there may be sugar in the blood and urine even if carbohydrates are entirely absent from the food. Such sugar must come either from proteids or fat, and the prevailing opinion is that it comes from the proteids; for in the first place diabetics do very well on fat, and in the next place only a little sugar could be produced even from a large amount of fat.* But it is clear that even in a case in which some carbohydrate is present in the food we cannot tell whether some in the blood may not have been derived from the tissues, if the patient has not completely lost his power of utilising ingested sugar.

The next point of interest is not nearly sufficiently grasped—namely, that many diabetics can utilise lævulose much better than dextrose. Many observers have noted this, and I have frequently observed it. It is said† that a diabetic patient soon loses his power to utilise lævulose, but I know he may retain it for a long while, and by examining the urine we can soon see when he is losing his power. This ability to utilise lævulose suggests that experiments might be made to see whether diabetics can utilise other sugars, *e. g.* pentose.

I have read many articles on diabetes which contain no reference to the extraordinary fact that, as was pointed out by Dr. Pavy many years ago, and which I have often observed, a diabetic patient who is on strict carbohydrate-free diet may, if a little carbohydrate is given, excrete far more sugar than that given. This is so important, and so little known, that I may be excused for quoting word for word from Dr. Pavy:‡ "The information supplied . . . goes to show that not only may starch and sugar ingested traverse the system unappropriated, but that their ingestion may act in such a way upon the complaint as to give rise to the elimination of more sugar than could be derived from the principles themselves. Thus after sugar had for some days been entirely absent from the urine under restriction to an animal

* Kraus, 'Lubarsch und Ostertages Ergebnisse,' Bd. ii, S. 615.

* Hesse, "Ueber Eiweissumsatz und Zuckerausscheidung des schweren Diabetekers," 'Zeitschrift für klinische Medicin,' Bd. xlv, Hfte. 4 and 5.

† Socin, 'Wie verhalten sich Diabeteker, etc.,' Dissert., Strassburg, 1894 (quoted by Bunge, 'Physiological and Pathological Chemistry,' second English edition, translated by F. A. Starling).

‡ Pavy, 'On Diabetes,' 2nd edition, 1869, p. 241.

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regimen, two ounces of ordinary bread per diem were for a couple of days allowed, and then the restricted diet again resumed. During the first day that the bread was taken the patient passed 117 grs. of sugar, and during the second 196 grs. The following day he passed 213 grs.; the next 571, then 236 grs., and lastly 92 grs. After this there was an absence of sugar. By the four ounces of bread the elimination of 1425 grs. of sugar was occasioned, which is more than could be derived from the bread itself, for I found . . . the whole of the solid matter in the bread consumed only amounted to 1200 grs., against the 1425 grs. of sugar discharged in the urine. Another point worthy of attention is that the elimination of sugar continued increasing for two days after the bread had been discontinued." Thus we see that the administration of sugar may act as a direct excitant of sugar-formation in the body (presumably from proteid); and if in any case some of the sugar taken is utilised, we have no means of knowing whether or not some of that in the urine is not derived from the tissues which have been excited to form sugar by the ingestion of carbohydrate. No explanation of the carbohydrate metabolism in diabetes will be satisfactory unless this fact is remembered.

Then, again, we want an explanation of the strange disappearance of sugar when coma comes on. This may be of great practical importance. Not long ago I saw, with Mr. E. F. White, of Putney, a patient who was not known to have diabetes. She became comatose in the course of influenza, and the first specimen of urine examined contained no sugar; but we were put on the right path by finding an abundance of aceto-acetic acid. Many years ago I tried giving one or two patients syrup, but it had no effect on the coma, and they did not live long enough for us to determine whether there was an increased output of sugar.

We much need information as to why the body fails to utilise dextrose. It has not lost the general power to oxidise; it can use fats; lactates in a diabetic are oxidised to carbonates, and benzol is oxidised to phenol. The only explanation open to us appears to be that usually offered—namely, that before the sugar can be oxidised it has to be prepared in some way, and the diabetic lacks the power to do this. Many symptoms—*e. g.* polyuria, the high specific gravity of the urine, and pruritus vulvæ—can obviously be explained by the presence of sugar, but, as previously mentioned, I think many symptoms are ascribed to hyperglycæmia on insufficient evidence; for instance, we are told* that these "results of hyperglycæmia consist chiefly in a striking tendency to infection and necrosis of the tissues." This may or may not be so, but, considering the many ways in which metabolism in diabetes is perverted, it appears rash to ascribe these results to the sugar. It is almost impossible to resist the temptation of believing that the wasting is due to the loss of energy in the urinary sugar. Many diabetics lose 500 calories of energy a day in their urine; and it is said that they attempt to make up for this by utilising their proteids and fats, and hence they waste. But, as Bunge points out, this is a teleological argument, and I think most of us feel that teleological arguments are dangerous. It would be a valuable observation to record the weight and nitrogenous excretion of a diabetic kept on a weighed ordinary diet, but absolutely at rest in bed, and therefore not requiring the energy lost in the urinary sugar. As sugar is chiefly consumed in the muscles, it is supposed by some that the fault in diabetes lies in them, and it has even been suggested that it is a good treatment for diabetics to do muscular work. Considering the loss of energy in the urinary sugar, this hardly seems likely, and it would be very valuable to have definite information.

In a debate on diabetes much attention must be given to the coma. It is even of greater importance than the glycosuria, for it is the commonest cause of death. At the present time the prevailing view is that the coma is in some way dependent upon the lævo-rotatory β-hydroxybutyric acid which is found in the blood of a patient in diabetic coma. It is excreted in the urine, and even as much as 119 grammes have been said to be excreted in a day. β-hydroxybutyric acid exists in three forms†—inactive, lævo-rotatory, and dextro-rotatory; in the body aceto-acetic acid and acetone are formed from it, and we usually infer its presence from the finding of one of these in the

* Edsall, 'Proceedings of the Pathological Society of Philadelphia,' October 1st, 1901, p. 232.

† McKenzie, "The Resolution of β-Hydroxybutyric Acid, etc.," 'Journal of Chemical Society,' November, 1902, p. 1402.

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urine. The right-handed acid is more easily decomposed in the body than the left-handed, which is the acid of diabetes. Two views prevail as to the way in which this acid acts to produce coma. One is that it or its precursor or its derivatives are directly toxic. Sternberg claims to have produced symptoms like those of diabetic coma by administering β-amidobutyric acid, which he regards as a precursor of β-hydroxybutyric acid; but it must be remembered, as Magnus-Levy * points out, that the amido-acids found in the body are of the alpha and not the beta group; and, further, I do not think that the animals who were poisoned with β-amidobutyric acid by either Sternberg or Grube appear from the written description to have had symptoms very like diabetic coma. Nor have these symptoms been satisfactorily produced by the administration of hydroxybutyric acid, aceto-acetic acid, or acetone; and, further, these bodies may be present without diabetic coma. The other view is that the hydroxybutyric acid acting merely as an acid diminishes the alkalinity of the blood, neutralising its sodium phosphate and carbonate, and hence the acid interferes with its power as a carrier of carbondioxide, and the carbondioxide consequently in the tissues is supposed to be the cause of the coma. About two facts there is no doubt. First, there is much oxybutyric acid in the blood, and its alkalinity is much reduced. By distilling the urine with strong sulphuric acid, crotonic acid is formed if oxybutyric acid is present; the presence of the acid has been in this way demonstrated by various observers, and also in the last two cases under my care. In both these the ammonia excreted in the urine was excessive; and Herter † points out that the calcium and other bases in the urine are much increased when oxybutyric acid is present, showing that the lessened alkalinity of the blood has led to the calling forth of these bases from the tissues, and by estimating the ammonia and bases in the urine he believes we can approximately learn the quantity of oxybutyric acid in the blood. As aceto-acetic acid is a derivative of oxybutyric acid, we usually roughly infer its presence by the ferric-chloride reaction. Secondly, there is no doubt that the carbondioxide content of diabetic blood is abnormally low, as shown for example by Kraus.* My friend Dr. Spriggs has kindly allowed me to give some results which he has obtained in the Physiological Laboratory at Guy's, working with Dr. Beddard and Dr. Pembrey. The gas estimations of the blood from cases of diabetic coma gave from 14 to 23 vols. of carbondioxide in 100 grms. of blood, instead of from 40 to 50, the figures found for normal venous blood. The alkalinity of the serum varied from 0·04 grm. sodium hydrate per cent. to 0·06, the normal being 0·1 to 0·15. The blood of two diabetics who were not comatose was examined, and it contained 24 and 33 vols. carbondioxide, so it was less than normal, but not so much less as in the comatose case. The carbondioxide content of the urine, too, was diminished. But it has always seemed to me a far cry to argue that because there is little carbondioxide in the blood, therefore it is retained in the tissues, for blood could probably carry away in simple solution enough carbondioxide to prevent toxæmia of the tissues by carbondioxide; and this is quite supported by what Dr. Spriggs tells me, for he and his fellow-experimenters found that blood from a case of diabetes can absorb carbondioxide about as well as ordinary blood; thus on passing carbondioxide through diabetic blood it took up 200 to 300 vols. per cent. of carbondioxide. This appears to show that the lack of CO_2 in the blood is not due to inability of the blood to take it up, but may be owing to a low tension of carbondioxide in the tissues in diabetes, and especially in diabetic coma. Therefore it appears that in diabetes there is a poison which has two effects—namely, the production of coma and the lessening of the carbondioxide in the tissues. What this poison is we do not know. That it merely acts as an acid is, I think, judging from experiments, doubtful; but there is considerable evidence that it is closely associated in some way with oxybutyric acid (although, as already mentioned, it is very doubtful if oxybutyric acid itself, its known derivatives, or amidobutyric acid are the poison), for oxybutyric acid is always abundantly present both before and even more during coma.† This view that the

* Magnus-Levy, 'Archiv f. experimentelle Pathologie und Pharmakologie,' Bd. xlv, Hfte. 5 and 6.

† Herter, 'Lectures on Chemical Pathology.'

* 'Lubarsch und Ostertag Ergebnisse, etc.,' 1895, Bd. ii.

† Herter, "Acid Intoxication of Diabetes in its Relation to Prognosis," 'Journal of Experimental Medicine,' vol. v, No. 6, 1901. Magnus-Levy, op. cit.

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poison is in some way closely allied to oxybutyric acid is strongly supported by the fact that sometimes coma is benefited by giving alkalies. I have seen distinct benefit follow, and Magnus-Levy records an excellent case; but as this treatment in no way destroys the poison or prevents its formation, at best only neutralising its acid properties, we must expect it often to fail, as, indeed, it does. It is interesting in this connection to learn that Dr. Spriggs did not find that the injection of alkalies increased the carbondioxide content of the blood in two fatal cases of diabetic coma, which is what we should expect if we are right in believing that the carbondioxide in the tissues is far below normal. I might, in passing, add that he found that in the last stages the administration of alkalies did not diminish the proportion of ammonia in the urine.

Much discussion has taken place as to the origin of the β-oxybutyric acid. It does not come from carbohydrates, for it may be present when they are withheld from the food and are absent from the urine. Until recently it was thought to come from proteids; now some suggest it is derived from fats, and again, Magnus-Levy suggests that it is formed by breaking down of both fats and proteids.* If it does come from proteids the metabolism which leads to it is probably different from that leading to sugar, for a strong ferric-chloride reaction may be present in the urine even after sugar has quite disappeared. I have noticed this in children, and if oxybutyric acid is derived from proteid we learn that at any rate not the whole of the abnormal proteid metabolism is to compensate for loss of energy in urinary sugar. Until we know the source of oxybutyric acid we shall probably not know how to avoid coma. Grube † believes it is likely to appear if butter is taken, but not after pork. This hardly seems likely, considering how well diabetics take butter. There seems no doubt that casts often appear in the urine in enormous numbers, especially before coma. It will be of great interest if this evening we can learn something of their origin and prognostic value.

The sugar and oxybutyric acid in the urine are not the only evidences of perverted metabolism, for sometimes the blood is of a peculiar salmon or strawberries-and-cream colour, a fact quite ignored by many who write on diabetes. Usually this blood is said to be fatty. Some analyses show a considerable amount of fat, and in some of the published cases * globules stained with osmic acid were present, but even then there was a quantity of granular matter; and a case I have been recently studying, and which I hope soon to publish, seems to show that the blood may be quite a pale strawberries-and-cream colour and yet contain no fat staining with osmic acid, but a quantity of granules which appear to be proteid precipitated by the presence of an ester of cholesterin with one of the fatty acids. The altered colour of the blood may be easily visible in the retinal arteries, and it may slowly pass away as the patient improves.

Turning to the morbid anatomy of composite diabetes, we know that the brain, spinal cord, and nerves show no naked-eye or histological changes. I have frequently examined these and the sympathetic ganglia with negative results.† But speaking of the nervous system reminds us that the explanation of the fact that the knee-jerk is absent in 50 per cent. of diabetics is difficult, considering how rare other symptoms of neuritis are. There are often striking changes in the pancreas. During fourteen years at Guy's Hospital, out of 6702 autopsies the pancreas was diseased to the naked eye in 142,‡ and in 19 of these the diseased pancreas was associated with diabetes. An examination of all the fatal cases of diabetes showed that in a quarter naked-eye disease of the pancreas was present. In 16 out of the 19 the gland was much atrophied, shrunken, and indurated; and this is, I think, the typical pancreas of diabetes. Of the remaining three, in one the gland was atrophied as a result of a pancreatic calculus; in one it was large and indurated. Hausemann, analysing the figures at the Berlin Pathological Institute, found in ten years disease of the pancreas with diabetes 40 times, and of these cases, in 36 the gland was atrophied. There appears no doubt that destruction of the islands of Langerhans is of great importance in connection with diabetes. A col-

* Op. cit.

† Grube, 'Zeitschrift für diatetische und physikalische Therapie,' Bd. vi, Hft. 2.

* Sanders and Hamilton, 'Edin. Med. Journ.,' July, 1879.

† "Diseases of the Sympathetic," 'Guy's Hospital Reports,' vol. xlvi.

‡ "Diseases of the Pancreas," 'Guy's Hospital Reports,' vol. liv.

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lection of cases by Steele * which has been recently published shows this. Of course it is well known that diseases which only partially destroy the pancreas do not cause diabetes. Three points on which we want information in this debate are—(1) Is alteration in the islands of Langerhans the essential feature of the pancreatic disease which causes diabetes? (2) Can we during life tell which cases have and which have not pancreatic disease? (3) What is the relationship of the internal pancreatic secretion to diabetes?

Passing to the subject of treatment, I need not urge the advantages of withholding carbohydrates from the food, for by doing so the patient gains strength and weight, and the excretion of sugar and oxybutyric acid diminishes, at any rate in most cases; but sometimes I have obtained an intense ferric-chloride reaction, even after weeks of strict diet, and even when the sugar has disappeared from the urine. It often seems to me that in many cases dieting has been too strict, and sometimes, at any rate, there is no harm in milk. In any case, I would suggest that when diet will not completely control the glycosuria the patient should be allowed just so much carbohydrate as will not increase the sugar in the urine, and when diet will control it he should be allowed carbohydrate food up to just below the point at which it leads to glycosuria. Lævulose is certainly the best carbohydrate; it may be given up to any quantity so long as it does not cause or increase the glycosuria. Inulin, which is the starch corresponding to lævulose, may also be given; it occurs in dahlia tubers, and these may be cooked and eaten like potatoes. There is a pernicious belief among some patients that toast may be taken instead of bread. I need hardly say this is not so. Although there are many reasons against alcohol, yet in a severe case advantage may be taken of its energy-yielding power, provided it is unsweetened. Many cases are probably not allowed enough fat. We should all be agreed that too sudden a deprivation of carbohydrates may lead to coma; but why this is we do not know, nor do we understand the undoubted and very important fact that coma is often brought on by exertion, worry, or mental anxiety. Unfortunately we are much in the dark as to the treatment of coma. As is shown by cases recorded by Fagge, the injection of normal saline solution will sometimes bring the patient out of coma, but he relapses again in a few minutes. Better still is the use of large quantities of alkali. If a patient feels a little drowsy it is a good thing to order 100 grains of bicarbonate of soda or 50 of the carbonate dissolved in a pint of milk, and to drink three or four such pints in the twenty-four hours. If the coma is deeper, a solution of sodium bicarbonate may be injected subcutaneously, or I have given it *per rectum*, but that is much less certain. If the case is at all severe, with an abundant ferric-chloride reaction but no coma, I am accustomed to order some 50 to 100 grains of bicarbonate of soda to be dissolved in milk and taken during the day. From cases I have watched I am certain this treatment is useful, and I think that it sometimes tides over a time till the good effect of dietetic treatment is felt. It often fails to save life, but then we must remember it does not stop the formation of the poison. It is evidence in support of this view that diabetics often appear to derive much benefit from alkaline waters. The bowels of these patients should be kept well open, for by that means we may eliminate some poison. Possibly the abdominal pain and vomiting which so often herald coma should be interpreted as an attempt to rid the body of poison by way of the gastro-intestinal tract. There appears no doubt that morphia and codein can both diminish the excretion of sugar; it will be of interest to hear why they are used less than they were. Aspirin is said to act beneficially in some cases, and I hope we may have some evidence for or against it. Dr. Garrod has told me of a child æt. 7, who on a fixed diet was excreting 30 grms. a day of sugar. She was put on 15 grains of aspirin a day, and the excretion of sugar fell, varying from 6 to 10 grms. After a week the aspirin was stopped, and the sugar excreted fell to 2 to 3 grms. a day. Aspirin itself being salicylate, will give a ferric-chloride reaction. I have tried feeding diabetics on pancreas,* and have injected liquor pancreaticus subcutaneously, but without success. The use of Prof. Starling's secretin does not appear to do any good. The treatment of the many associated conditions is beyond the scope of these remarks, but I cannot resist asking whether the number of cases of diabetic phthisis has not diminished of late years. I think it will be found that it has.

* 'American Journal of Medical Sciences,' July, 1902, p. 71.

* 'Brit. Med. Journ.,' March 4th, 1893.

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The last point to which I would direct attention is whether we are not under the word diabetes really describing more than one disease; for example, are some cases due to disease of the pancreas and some not? Are those that have so-called fatty blood different from those that have not? Are those in which glycogen is present in the liver different from those in which it is absent?

Dr. PAVY: Mr. President and Gentlemen,—I have listened with much interest to the able address that has been given to us by my colleague Dr. Hale White. But different minds approach a subject from different directions, and my mind has been concentrated upon diabetic glycosuria and non-diabetic glycosuria,—that is, upon the printed sheet before us, the distinction between the two. And the first point in drawing a distinction between the diabetic and non-diabetic glycosuria is to decide what constitutes diabetic glycosuria. We must first of all know what diabetic glycosuria really is before we can attempt to assign a position to non-diabetic glycosuria. Now the broad facts before us are that a diabetic subject differs from a healthy subject in this respect, or in these respects: the healthy subject takes carbohydrate food, and all he knows after taking this food is the benefit which he has derived from it. The food does not show itself as sugar in the urine, nor does it increase the amount of sugar in the blood. And the amount of sugar in the blood remains pretty constant no matter what the diet—taking an ordinary diet—may be, and the urine reflects the character of the blood. It is an index of the character of the blood as regards the sugar. There is a certain amount of sugar naturally existing in the blood, and there is a certain amount of sugar naturally existing in the urine. Increase the amount of sugar in the blood and you immediately increase the amount of sugar in the urine. We are in reality all glycosurics, not only ourselves but the lower animals; but only glycosurics to quite a small extent. And there is no doubt about being able to show the presence of sugar in normal urine. I do not rely upon the copper test, but there are other means. To-day I have seen some crystals of glucosazone which have been shown me by my assistant, obtained from his own urine, and he congratulates himself that he is not a diabetic; he congratulates himself that he belongs to the category of healthy persons in this respect. And by means of the fermentation test the presence of sugar is also susceptible of demonstration. I think it may be looked upon as a demonstrable fact that sugar is present in healthy urine, so that diabetes only varies as regards degree. I said that in the healthy subject the carbohydrate food is taken, and this does not increase the amount of sugar in the blood, so that the sugar in the urine comes from that constant sugar which is present in the blood. Now in diabetes the person is fed with carbohydrate food, and, in an ordinary case, in proportion as carbohydrate food is given, so is sugar present in the urine. The carbohydrate of the food can no longer be dealt with as it is in the healthy subject. We believe that in the healthy subject it is in part converted into fat and in part synthesised into molecules which can traverse the blood without escaping with the urine. Sugar consists of such small molecules that as it is passing through the glomeruli of the kidney it filters away in the same manner as water, as urea, or as saline matter. The molecules of sugar are so small that it runs through this filter which the glomeruli will represent. You cannot keep it in the blood. So that sugar reaching the blood cannot do otherwise than pass off with the urine, and thus if sugar reach the blood it will be sure to reach the urine, whilst the proper object of Nature is to keep it from escaping with the urine. Now by the process of assimilation the sugar is built up into larger sized molecules. We know that it is built up into glycogen. What is glycogen? It is nothing more than an extensive aggregation of, you may say, sugar molecules, with, at the same time, a certain amount of dehydration. You have molecules aggregated together, or, in other words, synthesised or built up into this large molecule, which is not susceptible of escape by diffusion from the position which it occupies. Or the sugar may be incorporated into proteid, and in the proteid molecule it can circulate freely without any danger of escaping with the urine. And there is no doubt that the sugar which is derived from the food passes by the process of assimilation into large-sized molecules, which can circulate through the body without running off with the urine, as the smaller sized sugar molecule does. In diabetes there is a want of this assimilative power; the power no longer exists of putting the carbohydrate of the food into a position to be retainable in the body. If taken with the food it

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reaches the blood as sugar and then runs off through the glomeruli of the kidney. In the diabetic there is a want of this assimilative power over carbohydrate matter, and hence, the carbohydrate matter of the food reaching the blood as sugar, the sugar which reaches the blood runs off through the glomeruli into the urine. In what is called the alimentary form of diabetes, to which Dr. Hale White in his address has alluded, there is simply this defect existing. It is simply that the carbohydrate which is taken with the food does not become assimilated, but reaches the blood as sugar and then passes off with the urine. Well, that is one source of the urinary sugar in diabetes,—the non-assimilated carbohydrate food. But we may have sugar reaching the urine from the breaking down of proteid which had been formed previously with the concurrence of sugar. And this is associated with what I have called the "composite" form of the disease. Proteid tissue breaking down, sugar is cleaved off from it, and the sugar reaches the urine. Thus we may have sugar in the diabetic from two sources—from the non-assimilated carbohydrate of the food, and from the breaking down of proteid matter. The former condition may exist alone, giving us the "alimentary" form of the disease. The latter, however, does not exist unassociated with the former; hence the term of "composite" applied to it. As in all cases of diabetes the former condition exists, we may give this as a definition of the disease: "A more or less persistent defective assimilative power over carbohydrate of food resulting in glycosuria." Now if we approach the consideration of diabetic and non-diabetic glycosuria from this standpoint, we can see into which category a particular case should fall. It may first of all be said that apart from disease anyone may be rendered glycosuric in consequence of our assimilative power over carbohydrate not being unlimited. Under ordinary circumstances a person does not get sugar in his urine from his food, but under unusual circumstances—under an undue ingestion of sugar—he may. Any person may be rendered markedly glycosuric by means of an undue amount of carbohydrate taken. Now this class of case will not fall into the category of diabetes, because it does not involve defect of assimilative power. In my definition I have said there must be a defective assimilative power, and here we have not the question of defect to deal with. The sugar in the urine results not from a defective assimilative power, but from carbohydrate having been taken to a sufficient extent to outstrip, as it were, the natural power existing. The glycosuria is due to abnormality as regards the amount of carbohydrate that has been taken, and not to abnormality as regards the assimilative power existing, so that this kind of case will not fall into the category of diabetic glycosuria; you have the glycosuria, but it is not diabetic glycosuria. In relation to diabetic glycosuria the widest variation is noticeable in the assimilative power existing. In some instances there may be only so slight a diminution of the power that there is no sugar in the urine under an ordinary mode of feeding. There is sugar in the urine, it is true, after taking an amount of carbohydrate that can be taken by a perfectly healthy person without passing sugar; but there is not much distinction, and in differentiating cases of this kind testing with food requires to be had recourse to. They are border-line cases. Sometimes there is sugar discoverable in the urine, at other times not, and the condition is most puzzling to medical practitioners. The patient goes to one medical practitioner, his urine is examined, and sugar is found. He goes afterwards to another medical practitioner, the urine is examined, and it is suggested that some error must have existed, as no sugar is present. These cases entirely depend upon the amount of carbohydrate which happens to have been previously taken. When the quantity is only moderate or is not in great excess there is no sugar noticeable in the urine, but at other times there is. Cases of this kind are often cropping up at insurance offices. A person applies for insurance; he is not aware of there being anything the matter with him, and at the request of the medical examiner he passes urine for examination. It is examined, and sugar is found to be present. Now is this to be considered as a diabetic or non-diabetic glycosuria? The person is taking ordinary food, and simply ordinary food, without any particular excess of carbohydrate. Have we here diabetic or non-diabetic glycosuria? Testing with food will decide the point. If you give to such a person a large amount of carbohydrate, if there is any weakness of assimilative power existing the evidence of that weakness will be brought out. Some time ago a person, in connection with whose condition

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different opinions had been given, was sent to me by an insurance office to be examined. The urine which he passed for examination a few hours after ordinary food for breakfast, including bread and toast, was found to be free from sugar. To know whether he really was a diabetic or not I subjected him to a test meal, consisting of bread and butter and marmalade, and this was accompanied by a glass of sherry. He took this meal in my house, so I was quite sure that the carbohydrate would be partaken of. In a couple of hours' time he came back, and the urine passed was loaded with sugar. I had no hesitation in saying that this person was the subject of a mild form of diabetes. In reference to mild glycosuria I may quote the following case, which is of much interest on account of the length of time it has extended over:—As far back as thirty years ago a patient came to me; his case had been diagnosed as one of diabetes, and he had been three months under treatment. The urine first examined by me only contained traces of sugar. He was under close observation for a week or ten days, and the outside amount of sugar that I found was 10 per 1000. The sugar afterwards disappeared and the patient returned to an ordinary diet. I have kept in touch with him ever since, seeing him perhaps once in two years, perhaps once in three years, or now and then every year. The last time I saw him was a few weeks ago. Now for a number of years—namely, until the year 1899—I never found any sugar in the urine, excepting at the commencement, and I was inclined to think that his case was not at the beginning one of diabetes. In 1899 sugar appeared, and the issue now is that he is obliged to keep to a comparatively restricted diet to keep his urine free from sugar. Looking at the whole history of this case, I think we must conclude that at the beginning it was really one of diabetes. It is an interesting circumstance that the son of this patient, a boy of nine years of age, in 1900, I think, was under Dr. Gee's care, and Dr. Gee found sugar in his urine. I have since seen this boy, and, as in the case of his father originally, his diabetes at present is of quite a mild type. I may cite another case, that of a lady who is only to a very mild extent glycosuric. I first saw her ten years ago, and she was then 28 years of age. The largest amount of sugar that I have at any time found in her urine has been 20 per 1000, and latterly she has been able to take nearly an ordinary diet with only traces of sugar discoverable in the urine. In this case there is also a family history of the disease, the father being glycosuric. Now cases of this kind I should consider as falling into the category of diabetes, but a very mild form of it. With them experience shows that the condition of the patient with respect to assimilative power may vary at different times. In the case of the patient to whom I first alluded, at one time it may be said that he was the subject of a mild form of diabetes, and this he recovered from so that he could afterwards live like an ordinary person without having sugar in his urine. We are not to look upon the condition of a patient as stereotyped or absolutely fixed, but the condition may vary from time to time, and a condition that exists at one time may be less favourable to the assimilation of carbohydrate than at another time. A striking case of this kind fell under my observation a few years ago. She has been for some years a comparatively mild glycosuric; if she takes much starchy food she has sugar in her urine. But with moderate restriction she can keep her urine free from sugar. She became the subject of a severe attack of pneumonia, and through the illness sugar was, apart from food to account for it, persistently present in the urine; and, further, the condition passed on into the "composite" type of diabetes. There were diacetic acid and oxybutyric acid present in the urine, and it seemed at one time as if coma were threatening. The patient recovered from her pneumonia and has since reverted to her original condition,—that is to say, one of mild glycosuria without the acetone series of products. So we must not look upon these persons as existing in a kind of fixed or stereotyped condition, but we must consider that the condition may vary, and that at one time the condition may be more favourable to the assimilation of carbohydrate than at another. At one time, for instance, a given amount of carbohydrate may throw sugar into the urine, which it may not do at another. Dr. Hale White has drawn attention to a case which was published by me many years ago, showing the effect of starchy food in giving rise to the elimination of a larger amount of sugar than could be derived from the starchy food taken. This result is in harmony with what may be observed in the progress of an ordinary case. A person becomes

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the subject of diabetes, and we will assume that its existence is not known; he goes on for some time, and progressively finds that he is growing worse. Supposing it to be a case which has begun mildly and then gradually advanced, the train of phenomena may stand as follows:—At the mild beginning the patient could take a considerable amount of carbohydrate without passing sugar, but as time goes on he begins to pass sugar on it, and then passes more and more upon the same carbohydrate. The case becomes gradually more aggravated; in other words, the assimilative power becomes more and more diminished, and the amount of carbohydrate which would only throw a little sugar into the urine at the commencement will later on be found to lead to the passage of a large amount. The case gradually grows as regards the defect in the assimilative power. Exactly the reverse occurs under proper treatment. A patient presents himself who is the subject of an amenable form of diabetes, and he is put upon the appropriate treatment. Perhaps under the restricted diet the sugar may not at first be susceptible of complete removal from the urine, but by-and-by it disappears. The case goes on, the patient meanwhile giving strict attention to dietetic management, and after the lapse of time, which cannot be foretold—in one case it may be two or three months, in another several months, in another it may not be for a year or two,—but by-and-by indications set in of a returning assimilative power. The indications are these: as long as the restricted diet is required from want of assimilative power the patient maintains his weight, maintains his strength, and feels in himself perfectly well. But with the return of assimilative power he experiences a sense of want and an epigastric sinking. He also feels weak and loses weight. You now give this patient a small quantity of starchy food. My usual plan is to begin with three ounces of wheaten bread per diem in one-ounce portions at each of the three principal meals. It is found that this allowance of bread throws no sugar into the urine, and the patient feels relieved at once and his weight goes up. You go on for a little time, and by-and-by you have a return of the former symptoms—a feeling of want and a drop in weight, and so on. This is to be met by an increase in the amount of carbohydrate given, and in this way I have frequently advanced up to seven, eight, and nine, and sometimes to twelve ounces of bread a day without bringing back the sugar. A patient may previously have been in an extremely diabetic state and passed sugar, and even passed sugar for a while upon a strict diet, but now he is taking a considerable amount of starchy food and is not passing sugar. The starchy food which is now taken is properly assimilated, and goes to the good of the system instead of running off as waste material with the urine. Of course you have absolute proof here that the condition of the system has changed. There is now a return of the assimilative power over carbohydrate to a significant extent. As a rule in these cases they do not regain their complete power; some do. Some are able to take ordinary diet without passing sugar upon it. But the usual event, according to my experience, is that there is a return of assimilative power to a certain, but only to a certain point, which varies in different cases. I bring forward these cases to show that we must not only look at the question from the aspect of food, but also from that of the condition of the system. The condition as regards the system varies, and with the variation there may be an increase or a decrease of the assimilative power; in other words, diabetes may grow worse or it may grow better.

A patient whom I first saw on May 30th, 1881, came to me for diabetes which had just previously been recognised. On a strict diet there was no sugar in the urine, but test diet brought out 57 per 1000 of sugar. Subsequently the patient was able to return to the ordinary diet without passing sugar. Another patient, whom I saw for the first time on February 15th, 1886. His urine then contained no less than 91 per 1000 of sugar. He was put upon the restricted diet. Four days later the night specimen of urine contained 19 per 1000, and the morning specimen 6 per 1000. The report for February 27th is that there was no sugar in the urine. In 1887 the patient returned to bread and potatoes without bringing a return of sugar. In 1888 the patient was taking an ordinary diet, and there was no sugar in the urine. December 4th, 1890, the report runs that the patient was taking any kind of food without any voidance of sugar. The conditions within had thus undergone a material change in the right direction.

The inference to be drawn from what I have said is that the presence of only a slight amount of sugar has nothing to do with the classification of glycosuria into diabetic and non-diabetic groups. Non-diabetic glycosuria I would speak of as independent of the question of carbohydrate assimilation. It arises from conditions unconnected with the character of the food. Obstruction of the breathing, for instance, as in asphyxia, may give rise to glycosuria, but this does not fall within the definition which I gave just now of diabetic glycosuria. In whooping-cough there may be sugar in

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the urine as a result of violent paroxysms of coughing. In connection with coma and convulsions sugar may happen to be found in the urine. One sees from time to time reports of these cases in the journals, where they are put down to diabetes. I remember some time ago seeing a report in which the condition was spoken of as diabetic coma. There had been no symptoms previously, and altogether the case read like one of ordinary apoplexy, leading, as is known to occur, to the incidental presence of a certain amount of sugar in the urine. Presumably the result arises from implication of the breathing. An anæsthetic may occasion a certain amount of glycosuria. You may have sugar in the urine in connection with lactation and constituting lactosuria. Further, glycosuria is met with as a result of poisoning by carbon monoxide. All these conditions I would classify as instances of non-diabetic glycosuria. In none of them can it be said that defective assimilation of carbohydrate is the source of the glycosuria. Probably an abnormal passage of glycogen into sugar is the operating factor. Glycosuria is sometimes met with in association with exophthalmic goitre. I have seen some few cases of the kind, and I am left somewhat in doubt as to how to regard them. Usually the amount of sugar is not large, and it may vary without the variation being definitely traceable to food, although I have known that influence has been exerted by food. In looking at these cases I have considered that the vasomotor condition which is leading to a dilatation of the vessels of the thyroid may be extended so as to more or less implicate the vessels of the chylopoietic viscera, and thus produce an effect of a comparable nature to that following Bernard's puncture of the medulla oblongata. In another class of case that is from time to time come across, sugar may happen to be incidentally found upon one occasion and not again, or if again, only temporarily in an erratic way. A patient whom I saw May 20th, 1890, and who had come to me as the subject of renal calculus, passed, at the time of consultation, urine for examination which reacted with Fehling's solution and was found to contain 8 per 1000 of sugar. No dieting was resorted to, and upon subsequent occasions there was an absence of sugar. Again quite recently this anomalous instance was come across. A proposer for life assurance at the Clerical, Medical, and General Office was examined by Sir Douglas Powell in the early part of the afternoon after an ordinary lunch, at which lager beer and a liqueur glass of Kümmel were taken. The urine passed for examination, I was afterwards informed, was of specific gravity 1005, and gave a pronounced reaction of sugar. The urine examined at a pharmaceutical establishment later in the afternoon was free from sugar. Some urine was passed for my examination on the following day at noon after an ordinary breakfast, into which marmalade entered, and was of specific gravity 1021 and free from sugar. I desired that carbohydrate food should be partaken of freely with marmalade plentifully next morning at breakfast. The noon urine afterwards was of specific gravity 1018 and free from sugar. It happened that the proposer dropped in at the insurance office after lunching in the same way as he had done before, and Sir Douglas Powell being there, he was asked to pass more urine for examination. This proved to be in the same condition as the first, and as some was sent to me for my examination I can give a detailed report of it. It was of specific gravity 1006, and threw down a neat precipitate of red oxide on being tested with Fehling's solution. A small quantity of sugar will do this in a dilute urine, when the same amount in a more concentrated specimen may only give a more or less indefinite reaction. The quantity indicated by the copper test was 4·19 per 1000, and by the polarimeter 4·2 per 1000. Good glucosazone crystals were obtained on treating with phenyl-hydrazine. Three days later, night, morning, and 2.15 p.m. specimens were sent from the country for examination and were all found free from sugar, and two days subsequently I saw the proposer again. The 11.30 a.m. urine was of specific gravity 1023 and free from sugar. It was now suggested that the original lunch should be taken, and that an early afternoon visit should be made to me instead of to the insurance office. At 2.15 urine amounting to about 100 c.c. was passed, having a specific gravity 1015, and containing 5 per 1000 of sugar. At 3.30 p.m. urine was again passed, which this time amounted to 700 c.c. (℥xxiv). Its specific gravity was 1003, and it contained 2·6 per 1000 of sugar. On the following day, at my suggestion, on account of glycosuria being recorded to have been noticed amongst Germans after free indulgence in lager beer, lunch was taken with the Kümmel afterwards, but with the substitution of whisky and soda for the lager beer. The urine afterwards voided was of specific gravity 1005, but this time free from sugar.

In another insurance office case also recently falling under my notice, sugar was found by the medical examiner in the urine of the applicant without any history to lead to the suspicion of diabetes. Some of the urine was sent to me for examination, and I found its specific gravity to be 1019, and that it contained 12 per 1000 of sugar. I saw the applicant the following afternoon, and after a lunch which constituted a good test meal her urine was free from sugar. Such cases as these from time to time present themselves, and the point to be determined is whether they are incipient cases of diabetic or whether they are instances of non-diabetic glycosuria. The food test is our great resource for arriving at a conclusion.

(*To be continued.*)

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THE RECOGNITION OF MASTOID DISEASE.*

By HERBERT W. CARSON, F.R.C.S.Eng.,

Assistant Surgeon, and Surgeon Ear and Throat Department, Tottenham Hospital.

After a short anatomical survey of the mastoid antrum, especially with reference to its relation with (*a*) the surface of the mastoid, to explain the occurrence or absence of periostitis; (*b*) the external meatus, to explain the presence of granulations or drooping of the posterior superior quadrant; (*c*) the middle ear and facial nerve; and (*d*) the cranial cavity and lateral sinus, the lecturer continued:

Mastoid disease may present itself either as an acute disease associated with acute middle ear suppuration, or as a more gradual process associated with chronic middle ear suppuration. I do not here include mastoiditis occurring independently of middle ear mischief, as such cases must be very rare indeed. When the mastoid participates in an acute middle ear suppuration it will generally occur as a sequel of scarlet fever, diphtheria, or much more commonly of influenza. In these cases pain radiating over the side of the head and face, and tenderness on pressure over the mastoid, are the prominent symptoms. The membrana tympani may be imperforate and bulging, and there will in most cases be a temperature of 100° or so. Do not expect swelling or redness over the mastoid, prominence of the auricle, or profuse discharge. The antrum is too deeply placed in adults for the periosteum to be early affected, and, unless the membrane is perforated, discharge will, of course, be absent.

In my experience it is much more common for patients to give a history of ear trouble of some standing, with varying discharge and occasional pain; and in these cases the condition is one of an exacerbation of a chronic mastoiditis which gave little inconvenience in the past, but is brought into prominence as the result of exposure or catarrh. And it is a very important fact to remember that mastoid disease may exist for years without giving rise to sufficiently severe symptoms to cause the patient to seek advice, and yet that these very cases may suddenly take a very acute course, "flare up," as it were, and lead on to the most serious complications. The diagnosis of some of these dormant cases is extremely difficult, owing to the vagueness of the symptoms, but if you ascertain that a patient with long-standing middle ear suppuration is the subject of occasional periods of malaise, attacks of vertigo or nausea, or that he has headaches relieved by the onset of discharge from the ear, or occasional pain or feeling of weight in the mastoid, —these are cases that you must view with great suspicion, and in such cases you would certainly be justified in warning the patient against travelling far from home or skilled advice.

In making your diagnosis of mastoid disease you will find you must rely much more on physical examination (objective symptoms) than on the history and subjective symptoms. Still, it is well to go carefully into the history of these cases, especially with regard to the length of time the discharge has existed, the constancy or otherwise of the discharge, the occurrence of headaches, and attacks of malaise, giddiness, or nausea. Find out about previous illnesses, remembering the fairly frequent incidence of mastoiditis after influenza. It is well also to inquire into the family history, for tubercle is responsible for a very insidious and painless form of mastoid disease, and complicated forms of ear disease occasionally occur in more than one member of the same family.

The points to which attention must be directed in one's examination are—

(1) The general condition of the patient.

(2) The external examination of the mastoid.

(3) The examination of the external auditory meatus.

(4) The examination of the membrana tympani, and if that is perforated, of the tympanic cavity and its contents.

1. *General condition.*—In the absence of complications, and in the less acute forms of the disease, the general condition does not differ materially from the normal. If there has been pain or even long-continued uneasiness, the patient may show the effect of sleepless nights, and in the event of an acute exacerbation there will probably be a rise of temperature to 100°—102°.

2. *The external examination of the mastoid.*—We must not expect mastoid disease to be invariably associated with redness, œdema, or swelling

* An address delivered before the North-East London Clinical Society.

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over the mastoid. While we may admit that in a certain proportion of cases these signs are present, and that if present they are a very valuable diagnostic sign, in the great majority of cases inspection of the mastoid in adults is negative. In children, however, owing to the comparatively superficial situation of the antrum, redness and swelling are much more common, and may be expected in the majority of cases. When swelling is present it will be confined to the mastoid, not obliterating the furrow between the mastoid and the pinna. If pus forms, the ear projects, and later on the furrow will disappear. In some neglected cases in children, an actual sinus, leading directly down from the surface of the mastoid to the antrum, may be present, but, of course, the diagnosis of such a case would give no difficulty. Palpation, which should always be done simultaneously on both sides, may give evidence of heat or tenderness. If the tenderness is due to mastoid disease it will be, in the absence of periostitis, confined to a small area directly over the antrum,—that is, just behind the posterior superior quadrant of the external meatal orifice, or over the anterior border of the mastoid, or at the tip. Remember that nervous patients may feel pain on pressure though the mastoid is not affected, and that a gland is situated a quarter of an inch behind the anterior border of the mastoid, and, if enlarged and tender, may simulate mastoid tenderness very closely. It is a good rule not to expect much tenderness, and to be suspicious of your diagnosis if tenderness is very acute. I have not found percussion help me at all, though it has been said that a duller note is elicited if the mastoid be diseased.

3. *The examination of the external auditory meatus.*—A good light is necessary, and in all cases where discharge exists the meatus must be thoroughly mopped out with cotton wool on a holder. Sometimes the meatus is more or less contracted owing to pre-existing eczema. This makes examination difficult, and is also of importance because of the possibility of retention of discharge being caused by the narrowed channel. As to the character of the discharge, neither fœtor nor profuseness is *per se* indicative of mastoid disease. If, however, the tubercle bacillus be found, or if cheesy particles, small cholesteatomatous masses, or minute portions of carious bone be detected in the discharge, there is almost certainly mastoid disease. The contour of the meatal walls should then be examined. Granulations are readily recognised, and when these are present one frequently obtains a history of occasional slight hæmorrhages from the ear. They may grow through a perforation in the membrane, or through the posterior meatal wall, in the latter case indicating disease of the limitrophic cells. If granulations recur after removal and adequate treatment, the diagnosis of caries and probable mastoid involvement is practically certain. Perhaps the most important diagnostic sign, as far as the meatus is concerned, is a drooping of the posterior superior meatal wall. It forms a prominence just external to the membrane; it may be large enough to almost hide the membrane; it is slightly more red than the normal wall, and is sometimes acutely tender to the probe. It is, I believe, never present unless the mastoid is diseased. It closely resembles a much more common condition, viz. a furuncle in the meatus, and it is very important, both from the point of view of prognosis and treatment, that these two conditions should be accurately differentiated. Difficulty arises from the fact that a furuncle, occurring, as it often does, in the course of a middle ear suppuration, may imitate mastoid disease very closely. Thus it causes pain, redness, and swelling over the mastoid, prominence of the auricle, and bulging of the meatal wall. The differential diagnosis may be made with certainty by observance of the following points:

(1) The furuncle gives rise to much more pain and tenderness than mastoid disease.

(2) The furuncle causes early swelling, and often pre-auricular glandular enlargement.

(3) If there be mastoid swelling, it tends, with a furuncle, to be diffuse, and to obliterate the furrow between the auricle and the mastoid, while in mastoid disease the swelling is more circumscribed and the furrow persists.

(4) The furuncle is always placed in the external half of the meatus, and is therefore never so deep, never so near the membrane, as the drooping due to mastoid disease. In addition, the furuncle is not confined to the posterior wall, and may be multiple.

(5) Furunculosis rarely, except in children, causes rise of temperature.

4. *Examination of the membrana tympani.*—This will almost always be found perforated. If it is not perforated it should be very closely examined

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with a view to the recognition of acute inflammation of the membrane (myringitis), or bulging due to retention of pus in the middle ear. Either condition, the latter especially, may give rise to very acute symptoms, resembling the subjective symptoms of mastoid disease very closely. The site of the perforation has been said to be of importance, it being stated that a perforation in the posterior superior quadrant, owing to its connection with disease of the incus, is the one most frequently associated with mastoid disease. If the perforation be large the condition of the inner tympanic wall can be observed, and carious bone searched for with a probe. It is possible sometimes to introduce a probe into the tympanic orifice of the aditus, and if bare bone is felt in this region the antrum is almost certainly diseased

Complications: Post-auricular abscess.—Let me again say that, except in children and in advanced disease, this condition is not common. If it occurs it is limited to the mastoid, and does not obliterate the furrow between the auricle and the mastoid until far advanced. It is fairly frequently associated with sufficient disease of the bone to cause a sinus leading from the surface to the antrum. In such cases diagnosis is not difficult, but considerable difficulty arises in cases where pus, burrowing through the mastoid, perforates near the tip, an abscess forming in the neck beneath the fascia. This is called "Bezold's mastoiditis," and may lead to a faulty diagnosis unless great care is exercised.

Facial paralysis.—The nerve is generally affected in the tympanic part of its course, but occasionally, as in a case recently under my care, the nerve is involved in its course through the mastoid. Facial paralysis due to middle ear disease is much more complete than paralysis of central origin. It may be due to a neuritis or to pressure from a sequestrum or granulation. It is a definite sign of advanced middle ear disease, and is an indication for operative treatment. The prognosis is not altogether bad in these cases, but the patient must be warned that recovery will be slow, and must be encouraged in every way to continue with massage and the battery, even though no apparent progress is made.

Extra-dural abscess may occur in the middle fossa as the result of caries of the roof of the tympanum, or in the posterior fossa as the result of mastoid caries involving the lateral sinus groove. Although it is said at times to follow a chronic course, in my experience it much more commonly gives rise to severe pain, rigors, restlessness, photophobia, nausea, and vomiting. The temperature remains high, and there may be retraction of the head and optic neuritis.

In *lateral sinus thrombosis* the clinical picture is very characteristic. The patient is obviously very ill. Headache and vomiting are early and well marked. The temperature rises rapidly to 104° or 105°, with fluctuations of a remittent type. The pulse is small, rapid and thready. Rigors occur early, are often repeated, and increase in frequency as the case proceeds. The tongue is dry and coated, the breath offensive. There is generally some mastoid swelling which may be marked in the region of the mastoid emissary vein, and extend into the neck, where pressure over the upper part of the internal jugular vein will cause intense pain. Eventually the patient sinks into a septicæmic state of the so-called typhoid type. Septic pneumonia may occur or secondary abscesses appear in the joints or viscera.

The last complication to which I wish to refer is *abscess of the brain.* The abscess may be situated either in the temporo-sphenoidal lobe of the cerebrum or in the cerebellum, the cerebellum being more frequently affected in adults, the cerebrum in children, for anatomical reasons, as I have stated before. The abscess may be the result of direct extension of disease, as is seen most commonly in temporo-sphenoidal abscess following caries of the tegmen tympani, or it may occur as the result of a pyæmic embolus. There may be a preliminary rigor, or the onset may be very insidious; in fact, many cases are reported of cerebral abscess having existed unrecognised for long periods. Both cerebral and cerebellar abscesses have certain diagnostic points in common, and differential diagnosis is difficult, if not impossible, unless focal symptoms (paralysis) be present. Common to both are fixed headache, slow cerebration, normal or subnormal temperature, slow pulse and respiration, and, in later stages, marked emaciation. In both cases there may also be vomiting and optic neuritis, though neither sign is constant. In *temporo-sphenoidal abscess* there may be paralysis of the opposite side owing to pressure on the Rolandic area across the fissure of Sylvius, affecting the face first, then the arm, the leg generally escaping. If on the left side, aphasia may occur. In addition, the third nerve on the side of the lesion may be paralysed, and the pupil of the eye of the same side become immobile. In *cerebellar abscess* there may be muscular rigidity affecting the limbs of the same side; conjugate deviation of the eyes to the opposite side; nystagmus, lateral or rotatory according as the abscess is in the lateral or central lobe; exaggerated knee-jerks on the same side; and vertigo with a tendency to fall towards the side opposite to the lesion. In cerebellar abscess, patients are said to have a tendency to lie on the side opposite to the abscess; in cerebral abscess to lie on the side of the abscess. Perhaps in any case the patient would be disinclined to lie on the diseased side if the mastoid were tender.

(The address was illustrated by notes of cases under the lecturer's care, illustrating the various complications.)

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A CLINICAL LECTURE

ON

INJURIES TO THE EYE.

Delivered at the Medical Graduates' College and Polyclinic, December 10th, 1902.

By H. E. JULER, F.R.C.S.,

Ophthalmic Surgeon to St. Mary's Hospital.

LADIES AND GENTLEMEN, — I read in 'Punch' this morning that there was about to be published a work called 'All my Eye' by the New Pupil, and I thought it must have some reference to my lecture, because this is certainly all my eye, and I am certainly a new pupil, for I have never been here before. So I hope you will receive me kindly.

My subject is injuries to the eye. Of course it is a very long subject, and it is not at all easy to bring it down to the practical limits of a lecture, but I thought I would try and get through it and make it as interesting as possible. I may commence by dividing up injuries of the eye into contusions and wounds.

Let us begin with contusions. Perhaps the best and most easily understood example of a simple contusion is a black eye. But there are many contusions without causing a black eye. A simple blow on the eye is a contusion. If there is such a contusion as will produce a black eye, the first thing is to find out whether the sight is affected. If the sight is not affected you may make your mind perfectly happy that there is no great harm done. The treatment of an ordinary black eye is perhaps too simple for me to refer to. Given a black eye and swollen eyelid, with more or less infiltration of surrounding parts, our first duty is to try the vision. And if you cannot get the eye open by fair means, you must do so somehow; you must endeavour to open the eyelids so as to ascertain what the vision is. If a patient has got as good vision in that eye as in the other, probably

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there are no complications. Secondly, if the vision is not good we have to suspect complications, and, of course, these complications are very numerous, almost too numerous to go into with any detail. If the vision is good you do not need to interfere further in the case, but if not, various things may have occurred to the injured eye. Everything depends upon the blow, upon its force and direction. One common form of trouble is hæmorrhage into the anterior chamber. That is an alarming condition. If a patient comes with his anterior chamber half full or full of blood, you will have to decide upon it and give a prognosis. The hæmorrhage may be in the vitreous, and not confined to the anterior chamber. If the hæmorrhage is not in the vitreous chamber it does not matter, because in the front part of the eye the blood will clear up without much trouble, perhaps in four days or so. But how are we to determine whether the hæmorrhage which we see is in the vitreous as well, or simply in the anterior chamber? You find it by means of reflected light. Have the patient in a darkened room and throw the light upon it by means of an ophthalmoscopic mirror. If only the anterior chamber is affected you will get reflection of light definitely. But if the hæmorrhage is present to any extent in the vitreous, if there is hæmorrhage from the ciliary body or choroid into the vitreous chamber, you will not get the reflected light at all; there will be darkness. Your prognosis must be guided greatly by the ophthalmoscopic condition found. Put some atropine into the eye so as to dilate the pupil, throw a beam of light into the eye and try to get a reflection from it. If the hæmorrhage is only in the anterior chamber the prognosis is good; but if it be in the vitreous the prognosis should be guarded and open, for blindness may probably result. A blow in the eye may be such that it ruptures the choroid, and the blow will expend itself on the posterior part of the eye. That is always attended by hæmorrhage into the vitreous chamber, and there may be no blood in the anterior chamber at all. If the hæmorrhage is not severe it can be seen in the vitreous by means of the ophthalmoscope.

Here is a diagram representing a very late condition resulting from a ruptured choroid, as seen with the ophthalmoscope. The rings are more or less concentric with the optic disc; and that is the usual position in which you find evidences of ruptured choroid. I must refer to the most important conditions of the choroid which result. One of them is called concussion choroiditis. A man who gets a less severe blow than that which I have just referred to, is liable to have the posterior part of his eye in a state of commotion. An inflammatory condition is produced called concussion choroiditis. It is a somewhat recent term. It looks something like retinitis pigmentosa, only occupying the posterior pole of the eye, and connected with the history of a blow. Detachment of the choroid may also occur when you get rupture, and the choroid may be separated from the sclera by the hæmorrhage which is produced, and that is very serious. The retina may be injured by this simple concussion, by a blow from a fist, or from a stick or stone, and even from a tennis ball. These concussions are very simple, or they are very severe. I remember the case of a doctor who was playing tennis when he received a blow from a tennis ball in the eye. He came to me with a central scotoma in the eye which was struck; there was quite a large area over which he could not see. Such patients are blind for both colour and form. He got quite well afterwards, but not for a long time. I take it that that was a simple case of commotion of the retina. Then you may have hæmorrhage into the retina from the same cause; or the retina may be split across; or there may be detachment. Detachment of the retina is very common, speaking from an ophthalmological point of view. Only this week I saw a young man who had detachment of the retina, and I saw one to-day in a woman. Neither of those patients when they came knew that they had had any blow in the eye, but both had typical detachment of the retina, presenting appearances very much like the diagram I show you. In this condition the lower part of the retina is thrown into rugæ, which are quite colourless. As far as one could tell, there was no history of injury, but careful inquiry made them remember a slight injury. The man had been hit by a ping-pong ball. I never knew until then that a ping-pong ball could produce such an injury; but he was a delicate youth, and he remembered the ball striking him in the eye. Any injury, or a fall, will produce the condition. A lady was knocked down by a bicycle, picked up unconscious, and taken to St. George's Hospital.

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She subsequently went home, and afterwards developed this trouble in the eye, no doubt from the shock she received. I believe her eye was struck at the same time. Instances of detachment of retina from concussion and contusion are very numerous. There is a late though rare condition — viz. "holes" in the macula; the macula shows small openings through which you can see the sclera. A blow on the eye may injure the optic nerve. The nerve may be severed entirely, or there may be hæmorrhage into the sheath. At the time of the accident you do not see this; you have nothing to guide you except the history and the blindness. Afterwards, compared with the normal fundus, there is a condition of atrophy of the nerve, and there is total blindness. These people never recover. The sclera may be next considered. It takes a very hard blow to rupture the sclera. I say without hesitation that if a blow is sufficient to rupture the sclera it is sufficient to destroy the eye; and if you find that the tension of the eye has become greatly reduced, and the vitreous is escaping through the wound, the eye is not worth saving. In fact, it is dangerous to keep such an eye.

Then there may be injuries to the lens by blows. Thus, there may be dislocation, either partial or complete. The lens depicted here is partly dislocated. Or the lens capsule might be ruptured partly or completely; generally this rupture takes place at the periphery, and so the nutrition of the lens is interfered with and cataract results. The bones about the eye are very likely to be fractured. It requires a great force to produce these fractures of the orbital wall; still, it does sometimes happen that in bad accidents the bones will be fractured, such as the shaft of a cart striking the eye. Then it is a more or less general surgical case, and is likely to be associated with intracranial concussion. The iris and ciliary body are liable to be affected. That is very interesting to us, because after a blow in the eye there may be a dilated pupil, or there may be a separation of the iris,—that is to say, an iridodialysis. Or there may be paralysis of the accommodation. When an iris is separated from its periphery it very seldom unites, in my experience. With the blood there it is difficult to see what the condition is; it will bleed profusely into the anterior chamber, and when the blood has been absorbed you will find the iris is separated from its periphery. The treatment is to put in atropine to dilate the pupil and keep the iris as far out as possible, so that it may get a chance of uniting again to its periphery, and trust to Nature to do it. There is nothing else to be done. But sometimes a blow in the eye leaves the pupil dilated, and that dilated pupil is always a condition which alarms the general practitioner. I have known many people who have been frightened because the doctor said it was a serious symptom. Undoubtedly it is if it indicates central disease, but inquiry about history of a blow should be made.

Next I pass to consider wounds. These I want to divide into penetrating and non-penetrating. Next I want to divide penetrating wounds into those which have no foreign body left in the eye, and those which have foreign bodies left there. I will pass as quickly as possible over the non-penetrating wounds. You may have the eyelid, or the sclera, or the cornea, or the conjunctiva affected. Wounds may be either punctured, incised, or lacerated, or due to burns or scalds. When the foreign bodies do not get into the eye, of course the condition is very much less dangerous than when they do. The chief trouble with these wounds of the eye is caused by the liability to septic infection. The eye is more or less like a cultivating chamber; it is a warm cavity—I mean the palpebral sac—with albuminous tears and saline fluid, and so it is a fine cultivating medium for organisms. The great danger of non-penetrating wounds is due to the fact that the surface of the eye is in a septic condition, and there is a liability to that septic condition getting into the eye. If there is a superficial wound or abrasion which is not visible to the naked eye, you know there is something of the sort present by the photophobia, or very likely to be; and if you take a solution of two per cent. of fluorescein, that will bring it up and make it evident to you, for instead of being a colourless transparent wound it will be of an opaque green, due to the stain getting below the level of the epithelium and into the substantia propria of the cornea. And this may be a good opportunity of referring to the eye as a lymphatic organ. There is a good deal of lymph circulation in the eye. For instance, the anterior chamber is a lymph space lined by epithelium, and it leads into the canal of Schlemm. The lens has

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a large lymph space at its periphery, in the region of the suspensory ligament (canal of Petit). And in the layers of the cornea there are lymph spaces; and inside the sclera, outside the choroid there is a lymph space going to the optic nerve. And undoubtedly the vitreous is full of lymph channels. They are not very well organised, but there is a free lymph circulation from the ciliary body for the nutrition of the vitreous. Thus the eye is a very apt place in which to get septic infection; and that is why, when we have a wound of any of these structures—eyelid, or sclera, or cornea—we have to be very careful, first of all, to remove any foreign bodies; secondly, to attend to the wound; and thirdly, to render it aseptic, as well as to cover it up and heal it.

I now want to deal with penetrating wounds. First of all, those without a foreign body in the eye. These are very common. Almost all one's specimens, apart from new growths of the eye, are specimens of injuries caused by such a thing as a pair of scissors, a knife, or a bradawl. First of all, there are the penetrating wounds of the cornea. These are very important, although often undetected. The cornea is a clear, transparent substance, and if the iris is not prolapsing a wound in it is apt to be overlooked. The fluorescein to which I have already referred will make it much more evident. We must be guided by the tension of the globe. If there is a wound in the cornea there is sure to be a more or less empty anterior chamber; the aqueous is almost sure to come out, and therefore the iris is much nearer the cornea than it should be, so that you will very likely find a hernia of the iris. It is wrong to shut up a wound of the cornea without careful examination. If a hernia of the iris exists it must be dealt with. How do we deal with it? I would say, first of all, cleanse the wound. If the iris is protruding, wash the surface of it and the whole palpebral sac with some mild disinfectant. We use perchloride of mercury. In general surgery it is used of a strength of 1 in 1000, but here for the eye, it is used of 1 in 5000 strength. If a stronger solution were used it would set up inflammatory trouble. Having cleansed it, get the iris back if possible; persuade it to go back if you can. If you cannot get it back by easy means you must cut it off—do an iridectomy. You must free that iris from the edge of the wound, or else you will have an anterior synechia formed, and there will be other troubles liable to be set up. Pull the iris a little way out and excise it with scissors, and then let the cut edges go back. The patient will, of course, have a coloboma afterwards, but that will not interfere very much with the vision after all; certainly it will interfere with it much less than if you left the iris clamped in the wound. It will slough off if you leave it there. A wound of the sclera is very important. It may be small and without any prolapse of the ciliary body. The question which must appeal to us when we meet with a wound of the sclera is whether it has entered the ciliary region or not. If a wound of the sclera occurs in the ciliary zone, it is very important and dangerous, for the reason that it is so liable to septic infection; hence this area is termed the "danger zone." Given a wound of the sclera, we first have to consider whether it is large or small. If it is large a great deal of vitreous will have come out in all probability, and there is no chance of saving the eye. The eye in such a case is very much disorganised, and the tension is decidedly reduced. If the wound is small we are justified in trying to save it. If the wound is in the danger zone we must speak with caution, and we try to save the eye. If the knife has gone into the ciliary body, this is so vascular that there is great liability to profuse hæmorrhage into the vitreous chamber, which is very dangerous; but if there is a clean wound I do not see why we should not make an effort to save the eye, especially if there has not been a large escape of vitreous. At the same time we have to be watchful of the other eye, giving meantime a guarded prognosis. The trouble is that you may not only get infection set up in that eye, but there may be an extension of the mischief known as sympathetic ophthalmitis in the other eye. This latter subject is too long for us to consider to-day; it is sufficient in itself for a whole lecture. When the lens is wounded by a penetrating instrument the wounded portion is sure to become opaque. The amount of opacity varies greatly with the size of the puncture. A small puncture of the lens capsule, as with a sewing needle, may only be followed by a slight opacity in the vicinity of the wound; whilst a large incision is pretty sure to be followed by swelling of the lens substance and opacity of an extensive nature. The severe swell-

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ing of the lens thus caused may set up glaucomatous tension in the injured eye, being characterised by severe pain, chemosis of the conjunctiva, and swelling of the eyelids. If such is the case the lens matter should be evacuated at once, as in ordinary cataract operation; otherwise the vision of the eye may be sacrificed from the glaucomatous attack. If no severe inflammatory symptoms set in immediately after a wound of the lens, it may be well simply to dilate the pupil with atropine and to wait until the eye has become quiet before dealing with the traumatic cataract thus formed.

I come now to penetrating wounds with a foreign body in them. I take this to be the most important part of what I have to say to-day. How shall we deal with them? First of all, one thinks of bodies which have flown into the eye from machinery or from a gun. We want to find out first, what is the nature of the wound; and secondly, we want to discover what is the nature of the foreign body, whether it is magnetic or non-magnetic; thirdly, we want to know the position of the foreign body, whether it is in the anterior chamber, the iris, the ciliary body, the lens, or the vitreous chamber. And next we must find out what was the date of the accident; for it must be remembered we have sometimes to deal with foreign bodies in the eye a fortnight or more after the accident happened. The wound will generally be a clean-cut one. For a foreign body to have got into the eye means it must have had some sharpness about it. The most common substances which fly into the eye are bits of steel, and the next in frequency are pellets of shot and gun-caps. You should first examine such a wound by means of focal illumination. Take a lens, and by means of it condense the light upon the eye and look at the wound. Try to make out from the history in which direction the particle came—whether from the side or from the front—and try to trace the object to its home. Possibly it may have gone through the eye. Sometimes it is very difficult to say whether a pellet has not gone in on one side and out at the other. Sometimes a shot will be found embedded in the fat of the orbit. If the piece of foreign body is in the anterior chamber you may be sure to see it, unless there is too much blood present. Also, if it is in the iris, you will be able to detect it. A piece of metal is often seen in the front part of the iris. If you dilate the pupil with atropine and the steel has lodged in the lens, you will be able to see it by focal illumination. If the body is in the vitreous, probably you will not see it by focal illumination; at any rate it would have to be very large for you to be able to do so. But with the ophthalmoscope, if there is not too much hæmorrhage, and unless it is too much in the anterior peripheral part of the vitreous, you will see it at the back of the eye. Often a foreign body may be seen floating in the vitreous, or sticking on the retina towards the posterior pole. Still, the body may be so covered up that you cannot see it either by focal illumination or by means of the ophthalmoscope. Then you must avail yourself of the more recent improvements—namely, those in skiagraphy. For instance, there is Mackenzie Davidson's instrument for localising a foreign body in the eye. He puts a piece of metal on the lower eyelid to constitute one known point, and then he has two cross copper bars close to the patient's head, and the patient's head is put into the X-ray instrument and held rigid, and then two stereoscopic photographs are taken from different positions, the lamp being moved through three centimetres between the two pictures. These two pictures when combined show the object as standing out in the exact position occupied by it. He also uses a localiser by which the co-ordinates of the position are measured and compared with a known fixed point, and thus the exact position and size of the foreign body to within one fiftieth of an inch are estimated. If you have a good skiagraph you can often see whether a foreign body is in the globe or not, without the localising apparatus. In the case of a recent accident, if you cannot see the foreign body, you may find evidence of the commotion it has made in the eye, and you can perhaps discern its track of entry. In such a case as that the skiagram comes in useful. You may get a case coming some time after the injury. The patient may be blind, or he may suspect that there is a foreign body in the eye. That is always indicated by certain symptoms. Whenever there is a foreign body in the eye—whether it be in the cornea, or anterior chamber, or iris, or ciliary body, or vitreous—you will almost always find redness of the circumcorneal zone and congestion of the conjunctiva. There are two instances of foreign body in the

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eye where there may not be much disturbance—namely, where a very small portion of material, such as aseptic metal, has entered the lens. In such an instance you may have very little trouble indeed. Secondly, when a clean piece of gun-cap, having entered the eye, rests against the retina. I have seen a case in which a piece of metal flattened itself on the fundus of the eye near a vessel. After it had been there for a time it shifted a little, and the only sign of its presence was that the patient had a scotoma in his field of vision corresponding to the position of that particular bit of metal. But such cases are very exceptional. There is generally sure to be congestion of the circumcorneal region when a foreign body has entered the eye. As a rule there is violent inflammation in the eye. If the foreign body takes any septic material into the eye there is sure to be inflammation of the iris, and ciliary body, and vitreous. One could relate a number of cases where, what seemed at first to be a very simple injury, a foreign body penetrated the eye and remained in it, septic infection was set up, and in a few days the eye was entirely lost, and its presence was dangerous to its fellow. One way of testing the presence of a magnetic body is to bring a strong electro-magnet near the eye. The effect of the magnet will cause pain. Haab's magnet, which is here shown, is the best and most powerful instrument. It has a cone-shaped projection, and the eye must be approximated to the apex of this cone, and so exposed to the magnetic action. A magnetic body in the eye is thus powerfully attracted in the direction of the magnet. If the body is not moved from its position it will cause pain; if moved it may pass from the vitreous cavity forwards, and be seen in the anterior chamber; or it may even pass through an opening in the cornea or the aperture of entrance, and so be removed from the eye. Other less powerful magnets are used which have nozzles of various sizes. The latter can be introduced into the eye and made to approximate the foreign body, either through the original wound or through another opening made for the purpose.

This removal of magnetic bodies from the eye by means of a magnet is the most interesting recent development of ophthalmic surgery. Its results are frequently of the most brilliant and satisfactory nature. The chief practical point about this method of removal is to make an opening in the eye in such a position as to favour the escape of the foreign body with the least possible disturbance of the organ.

Non-magnetic foreign bodies lodged within the eye must be removed at any cost, because if not removed that eye is sure to be lost from chronic or acute inflammation, or by shrinking, and it will threaten the other eye. If the non-magnetic substance is on the iris you will be tempted to take a small pair of forceps and grasp it with them, but this method is liable to failure, inasmuch as the iris so easily bleeds when touched, and the foreign body can no longer be seen; it is better to grasp the iris on one side of the body, and then on the other, making a sort of fold or cul-de-sac of the iris, and pull that part of the iris out through the opening you have made and do an iridectomy. If you are fortunate you will find you have the foreign body wrapped up in that fold of iris. The eye will not be much affected by the iridectomy after the foreign body is out. If it is in the lens you must leave it or not, according to the amount of trouble. It will set up cataract, either partial or complete. If the symptoms are not acute, if the swelling of the lens is not enough to set up glaucoma, operative measures may be postponed until a convenient time. When acute glaucomatous symptoms are produced the lens must be extracted at once, and, if possible, the foreign body with it—just as in ordinary extraction. It may be that the condition is so slight that the foreign body may remain there for the patient's lifetime. I have a patient with a piece of metal in his lens for which he seems to be none the worse. If the non-magnetic body is in the back of the eye you must go for it either through the point of entrance, which is not often useful, being generally in the front of the eye, or you make an incision through the sclera, choroid, and retina, and as near as possible to the ascertained position of the foreign body; through this aperture a pair of forceps or curette is introduced, and removal attempted.

If you cannot get the foreign body out, what are you to do? Shall you remove the eye or eviscerate the eyeball? There is a modern practice in favour of evisceration, and the introduction into the cavity of a glass ball, called Mules's operation. That is very nice as long as there is not much

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septic material, and if you watch the eye very carefully. The introduction of a glass ball in the eye makes a better appearance, and there is a good stump left, but the misfortune is that the operation is sometimes followed by sympathetic trouble. I show you a jelly preparation of an eye in which the globe was used, but in which sympathetic trouble supervened in the other eye, and the stump had to be removed. I am glad to say the eye made a complete recovery. You cannot too carefully keep in mind that an eye in which a foreign body is left must be lost, and that if it is not removed by operation at the time it will be very likely to set up sympathetic ophthalmitis in its fellow.

Gangrene following Operation on X-Ray Cases.—Dr. SAMUEL LLOYD considered with regard to a case of osteosarcoma that we should now revise some of the claims made a year ago concerning the X-ray treatment. Even the most enthusiastic X-ray workers had now recognised that such treatment did not seem to influence favourably the deeper growths. He had seen two cases of epithelioma in which the proliferation had gone on very much more rapidly since beginning the X-ray treatment, although the latter had been thoroughly carried out by experts. He had recently operated upon two cases that had been subjected to X-ray treatment for a considerable time. In one there had been no X-ray treatment for some weeks prior to operation; in the other case the X-ray treatment had been continued up to the time of operation. In the latter case, although the parts were brought together without tension, a gangrenous patch developed, and nearly covered the whole area that had been exposed to the X ray. At the time he had not attached very much importance to this, believing that it was due to some fault in the technique, but when he had subsequently observed a second case of the kind he had been led to ponder over the significance of the observation. The case was one of carcinoma of the breast, and while he obtained easily an abundant flap there developed subsequently a gangrenous patch covering the whole side of the chest.—*Medical Record*, April 4th, 1903.

DEBATE
ON
DIABETIC AND NON-DIABETIC GLYCOSURIA.

Held under the auspices of the Chelsea Clinical Society at the Jenner Institute of Preventive Medicine, March 10th and 17th, 1903.

Dr. C. C. Gibbes, President, in the Chair.

(*Continued from p.* 13.)

Dr. SAUNDBY: As the hour is late I will not trouble you with many remarks, but I would like to ask your attention for a short time to several points in connection with the subject under consideration. I propose to follow Dr. Pavy in the line he has taken, in discussing more particularly the subject which was set before us—namely, glycosuria, diabetic and non-diabetic. Although I have listened with the greatest pleasure to Dr. Hale White's admirable paper, I do not propose to follow him in the general question of the pathology of diabetes; I want more particularly to keep to the practical question of glycosuria. Now in the first place, this is the way I understand glycosuria: Dr. Pavy has told you that glycosuria is a matter of degree, that the normal man passes glucose in small quantities not appreciable by ordinary tests. I imagine we should be agreed in meaning by glycosuria that which is appreciated by clinical tests and our ordinary test is Fehling's solution, which I think is not such an unsatisfactory test as Dr. Hale White has suggested. I might say that, of course, one has to confirm it every now and then by some other means; but those cases are not very common, and it is always possible to reserve a specimen and confirm it by one of the other various means, which need not detain us now. That being so, I understand glycosuria in this way: the normal man is capable of assimilating a certain amount of carbohydrate, but, as Dr. Pavy has insisted over and over again, there is no fixed quantity. Men differ among themselves; some men tend to store up a great deal of carbohydrate in the form of fat; others burn it off. And then, again, this combustion of carbohydrate is more vigorous in young people than it is in those who are getting older. What we may call the sugar-destroying or sugar-burning function of the body diminishes as time goes on, and after middle age

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it certainly diminishes considerably. That is why it is such a common thing to meet with glycosuria in elderly people. Therefore a good deal of the glycosuria of elderly people is simply alimentary glycosuria,—that is to say, these people go on eating and drinking, perhaps more as they get older, because I think as we get more into years our appetites do not get less; we stay longer at the table and do not take so much exercise, and we are more likely to take an excessive quantity of carbohydrate food, and therefore to have a surplus which passes off in the urine. That is the alimentary glycosuria of elderly people. The next point is this: Dr. Pavy has rightly attempted to distinguish between non-diabetic glycosuria and diabetic glycosuria. As I understood him, he said that diabetic glycosuria was a glycosuria in which assimilation failed, and the non-diabetic was that in which assimilation was not at fault. I am not quite able to accept that definition, because I think in a given case it is difficult to know when assimilation is at fault. I would suggest a more practical, as it seems to me a more clinical distinction,—that is, when we know what the cause is, and especially when it is transient or removable, we should not call it diabetic glycosuria. Where we do not know the cause, or where the glycosuria persists, it is probably diabetes. For example, nothing has been more strongly impressed upon me of late years than the truth, which is not generally admitted, and has been denied by many great authorities upon diabetes, that alcohol is one of the commonest causes of glycosuria. Dr. Pavy's recent case was one in point. Perhaps Dr. Pavy would say that the glycosuria was due to the carbohydrates in the lager beer; but I have often seen the same effect produced by whisky. Dr. Williamson, of Manchester, who has written a good book on diabetes, is strongly convinced of the effect of alcohol in producing glycosuria. I have over and over again seen it produce temporary glycosuria. If you have a man who comes to you sodden with drink, almost in a state of delirium tremens, I think you may be tolerably certain that sugar will be found in his urine. It is true it may pass off in twenty-four hours. A man comes to be examined for life insurance who has dined heavily the night before, or has just lunched freely, and you find sugar in the urine. I remember a man coming to me—he was an officer of Inland Revenue who had been recently promoted, and some friends had given him a dinner to celebrate the affair. On the strength of his increased pay he wished to add to the amount of his life insurance, and he went to the insurance office to speak about it. He was asked to come in and be examined at once. He said he did not want to do so then, as he knew he had had a good deal of drink the night before, and he was not feeling quite up to the mark. However, he was persuaded to be examined, and sugar was found in his urine. He came to see me the next day, but the sugar had disappeared. That case occurred some years ago, before I had become convinced of the frequency with which alcohol produces glycosuria. Some of these cases become really diabetic. I had such a case not long ago under my care, in hospital. The patient had a brother who was diabetic. Three times this man came into the hospital, and twice went out very much better; but previously to each admission into the hospital, as he admitted, he had been taking drink freely. On the third occasion, which was at the end of last year, he developed coma and died. There are many other causes of temporary glycosuria besides alcohol. Some of those have been touched upon by Dr. Pavy, and therefore I will not go into them now. We often see glycosuria in association with boils and carbuncles. A colleague of mine, a surgeon, got what surgeons often get in connection with wound infections—a row of little boils up his arm. He was feeling out of sorts, and sugar was found in his urine. He came and told me about it, as it had lasted some time. I know him intimately, and I have examined his urine since, but the sugar completely disappeared in six weeks, and it has not returned. It was simply due, I feel sure, to the presence of these boils that I have mentioned. Mr. Spencer, of Westminster Hospital, has published a good deal upon this subject of glycosuria in connection with surgical wounds and infections; and there is no doubt that it occurs commonly. I have a case under care now—that of a nurse with wound infection, with glycosuria. It has lasted two months, and she had, when I examined her last, nearly 2 per cent. of sugar. She had not been dieted, as I had not thought it necessary, there being no other symptoms. However, as the glycosuria has persisted, I have restricted her carbohydrates.

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The next point I wish to allude to is the fact that all people do not react in the same way to all forms of carbohydrates. Not only do people differ very much in their individual capacity for assimilating the same carbohydrates, but the same man will react differently to different forms of carbohydrates; they will assimilate some better than others. That has been already alluded to to-night. You have been told that diabetics can assimilate lævulose, and in my experiments I have found that many diabetics can take one to one and a half ounces of lævulose daily without producing any increase in the amount of sugar excreted. But there are other things which are of more practical importance than that. Lævulose is rather expensive, and it does not after all help us very much as a food. Milk is much more important; it is a very valuable and generally popular article of diet, and it is a great thing to be able to know that most diabetics can assimilate definite small quantities of milk—half a pint to a pint—containing 200 to 400 grains of lactose, without producing any increase in the quantity of sugar excreted. But, while I say that that is the general rule, we must always watch the effect, and not trust to general rules. The whole twenty-four hours' urine must be collected and the total quantity of sugar estimated. As has been said, it is as absurd to speak of a diabetic as passing 5 per cent. of sugar as it is to say that a man's income is 3½ per cent. or 3 per cent. You want to know the actual quantity in grains of sugar excreted daily, and the patient is put upon a diet of known carbohydrate value. We can then easily see whether an additional half-pint of milk makes a notable difference in the excretion of sugar. Milk is a very important article of diet, which most diabetics—we cannot lay down an absolute rule in the matter—can take without increasing the quantity of sugar. Another common and popular article of diet is potato. Professor Mossé, of Toulouse, has been recently writing a great deal about potatoes and recommending their use in large quantities. It was Dujardin-Beaumetz who first preached the value and utility of potatoes in the treatment of diabetes; at least it was from his paper that I learnt of them. He pointed out that potatoes contain only a small amount of starch; that whereas bread contains about 50 per cent. of starch, potatoes contain 15 to 20 per cent. of starch. You may give a man a potato weighing 6 oz. and yet be certain that it will not contain more than 120 grs. of starch; and remember, a big potato like that is a very comfortable thing, which enables a man to get through a meal without troubling about toast or bread. Supposing that instead of this potato you gave him one single ounce of toast, it would contain from 300 to 400 grs. of starch. Not only is the potato much more suitable for the patient, but he will often prefer it. I have been in the habit for many years past, after determining the amount of sugar excreted in twenty-four hours on strict diet, of giving half a pint of milk and trying its effect. Then I give 6 oz. of potato daily. Mossé advocates as much as 1000 to 1500 grms. of potato daily. I do not usually go so far as this, but I give as much as 4 oz. of potato with each meal in some cases, and my experience shows that the plan is worth trying. Potatoes do not contain as much starch as gluten bread, and yet you do not hesitate to give gluten bread, which is much more expensive than potato. The following cases illustrate the value of potatoes:

Case 1.—John S—, æt. 21, had lost 17 lbs. in weight. Placed on strict diet. In 110 oz. of urine there were 1672 grs. of sugar. Diacetic acid was present and the ferric chloride reaction was given, but there was no albumen. I added 1½ lbs. of potatoes to his diet. His urine fell to 76 oz. and the specific gravity to 1032, and the total sugar was 1596 grs.; that is to say, it was less on that diet than it was on strict diet. The man went out of hospital against advice, after having been in only eleven days, but he had gained 5 lbs. in weight in the time. For a man who was only twenty-one, and a severe case, this was a fairly good result.

Case 2 was a woman æt. 48, who had been ill three months and had lost 2 st. in weight. She was suffering from pruritus vulvæ, thirst, polyuria, etc. On strict diet the urine fell to 34 oz. and the sugar disappeared completely; there was no diacetic acid. She took 1½ lbs. of potatoes daily, but the sugar did not return. Then I added 4½ oz. of toast—that is to say, 1½ oz. with each meal,—and still no sugar returned. It was evidently a mild case, and you may say she could take almost anything.

Case 3 was a man, Alfred C—, æt. 48. He had been in the hospital before with diabetes; he was emaciated, weighing only 7 st. 12 lbs. I ordered

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1½ lbs. of potatoes daily in addition to strict diet. His urine was 110 oz., specific gravity 1030, total sugar 2310 grs., which I thought was too large a quantity, so I put him back upon strict diet. On that the sugar fell to 634 grs. I then gave him 6 oz. of potatoes and half a pint of milk, and the sugar fell to 510 grs. He was given 4½ oz. of toast, and the sugar went up to 2155 grs. The toast was stopped and 12 oz. of potatoes substituted, and on that he passed only 880 grs. of sugar. Diacetic acid was present in this case throughout.

The first case was under observation too short a time to enable me to speak of the ultimate result, but so far as the experiment went it was not unfavourable. In the second case the potatoes answered admirably well. In the third, which was under treatment a month, the man could not take toast, but tolerated potatoes; he took 12 oz. of potatoes a day and passed only 880 grs. of sugar, as against 500 grs. upon strict diet and over 2000 grs. upon toast.

These cases are quoted as an encouragement to try the effect of potatoes as a substitute for bread. In gluten bread, which was used for so long for these cases, there is at least 25 per cent. of starch; it cannot be made without.

Dr. PAVY: Oh yes; you can get gluten bread containing 10 per cent. or less.

Dr. SAUNDBY: It was formerly made with at least 25 per cent. of starch. Mr. Callard now makes a starchless bread, but for a long time we had only gluten bread, which contained a large percentage of starch, sometimes as much as 40 per cent. Ebstein's bread is made by the addition of 25 per cent. of starch to aleuronat flour, and it is said that it is impossible to bake *gluten* flour with less than 25 per cent. of starch.

I have notes of many other points, but I will not trouble you with them to-night. Those I want to impress upon you are, first of all, that there are many cases of glycosuria which are not properly diabetic glycosuria, in which a cause can be found if you look for it; and one of the commonest which you may meet with in practice is alcohol. The second point is that people vary very much in their individual capacity for assimilating carbohydrates, and that if you wish to treat your cases successfully you must calculate their total output of sugar and not be content with percentages; that you must also calculate the total amount of carbohydrates which you are putting in as well as that which is going out. There is another point to which allusion has been made, and upon which I ought to say something,—the sugar which is eliminated is not wholly derived from the carbohydrate of the food. Sugar is formed from proteid; it is formed probably by splitting off from the proteid molecule or in some other way. In any case, a considerable amount of sugar is formed in the body in the process of breaking up albumen to form urea. A rough method of utilising this fact can be arrived at in this way: if you know the total urea you may double the quantity to get the amount of sugar derived from proteid; if your patient is passing 500 grs. of urea he has formed 1000 grs. of sugar in his body in the process of manufacturing the urea, so if you give him a diet which contains 1500 grs. of carbohydrate and he excretes 500 grs. of urea, you must add another 1000 grs., which will make 2500 grs. of sugar, which the patient will deal with somehow—burn it up, store it as glycogen or fat, or excrete it as glucose in his urine. It is very desirable to make this calculation, so as to see what your patient is doing. Another important practical point is to weigh your diabetic patients regularly. They need not be weighed too often—once a fortnight is enough. Do not encourage them to use a urinometer and be always testing the specific gravity of their urine; nor would I let anyone estimate sugar in his own case. I saw such a case the other day. He was an officer in the Army; he had supplied himself with the whole apparatus for estimating sugar, and had been testing his own urine every day, and was in great alarm whenever the sugar showed a slight increase, although the amount was so small that at most it was only 0·25 per cent.!

Dr. BRADFORD, F.R.S.: It will be impossible at this late hour to deal with the wide range of subjects which Dr. Hale White has brought before us. The debate this evening shows, I think, that we shall probably be all agreed in recognising that there is a non-diabetic glycosuria, and for my part I should think that that is a very considerable advance in our knowledge, because it is not so very long ago that the presence of sugar in the urine was looked upon as pathognomonic of diabetes. At a slightly later period than that it was thought that some of the cases which we now recognise as non-diabetic glycosuria were really due to erroneous

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observation and due to the presence of other reducing substances in the urine. At the present day we have, then, to deal with three great groups of cases : those in which there is another substance not sugar in the urine, but which is confounded with sugar ; those in which true sugar is present in the urine, and which we do not look upon as diabetes ; and those in which the sugar is present, and which we do look upon as diabetes.

The first of these groups does not come within our discussion this evening, and so one may dismiss that group by saying that the reducing substances other than sugar which are found in the urine occur normally, and also as a result of disease—as, for instance, uric acid, creatinin, and glycuronic acid. The fallacies they give rise to may be dependent upon the urine being excreted in a more concentrated form, as in the instances which arise owing to the presence of uric acid and urates being mistaken for sugar ; or, what is more important, they may be increased in amount owing to the ingestion of a number of substances. At the present time we are only acquainted with drugs, such as camphor, chloral, and carbolic acid, which will produce this effect ; but still it is possible that the action which we know to be associated with these drugs may also occur with other toxic substances the nature of which is not yet known.

Putting those aside, and passing to non-diabetic glycosuria, we are of course brought at once face to face with the problem which other speakers have raised as to how they are to be separated from the diabetic. I think we must admit that some of them cannot be separated with any degree of certainty from the slighter forms of what Dr. Pavy and others have called alimentary diabetes. There is one characteristic of them which, I think, has scarcely been alluded to sufficiently this evening, and which to my mind seems to be a very special characteristic of them. This is, that the glycosuria of non-diabetic cases surely has this very fundamental character, that it is transitory, that it is persistent only for a very short time, and, as I think Dr. Pavy or some other speaker this evening mentioned, that sometimes it is only present on one occasion. That is a very great characteristic of these cases of non-diabetic glycosuria. The mere amount of the sugar present I suppose we should all agree upon as being a very misleading criterion. In some of these cases there is a large amount of sugar, and in some whose subsequent course is that of genuine diabetes there is only a small amount of sugar present. So the quantity cannot be laid stress on, though the transitory character of the glycosuria can be.

With regard to the causes of this non-diabetic glycosuria, there is one which is very definite, and that is the glycosuria that occurs as a result of cerebral injuries and in cerebral diseases. It may not be very important clinically, but still it is probably the most definite that we are acquainted with, and especially the glycosuria associated with hæmorrhage in the region of the fourth ventricle, such as basilar aneurysms and other forms of cerebral lesions leading to hæmorrhage into the fourth ventricle. In considering this form of glycosuria I think we should bear in mind the fundamental experiment of the so-called diabetic puncture, which, by the way, may be looked upon as a rather unfortunate name for it, because it is not like the experimental diabetes that is produced by other measures, such as the removal of the pancreas. This experimental glycosuria is on all fours with the glycosuria that is seen in these cerebral diseases and cerebral injuries.

We shall, I think, also admit that there are certain people who suffer from non-diabetic glycosuria as a result of the ingestion of large quantities of carbohydrates. Whether a perfectly normal person can ever pass sugar as a result of mere ingestion of large quantities of carbohydrates I have not sufficient experience to say, but we know that very large quantities indeed of sugar have been taken by normal persons—enormous quantities—without any appearing in the urine. On the other hand, we have all probably met with patients presenting no signs of ill-health, and who only pass sugar as a result of taking large quantities of carbohydrate, and the whole thing disappears not by cutting off carbohydrates from the diet, but by bringing back the diet to a more or less reasonable character. These might certainly be described as cases of non-diabetic glycosuria, although perhaps it is difficult to draw any hard and fast line between these and the slighter forms of alimentary glycosuria. The point of distinction is that the glycosuria only appears as a result of the ingestion of extremely large quantities of carbohydrate material. There is also no question that glycosuria may be produced by a number of poisons, such as morphia

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and other poisons, quite apart from the production of glycuronic acid. I do not propose at this late hour to quote cases to you, but there is one I want to draw attention to particularly, in case any one here can throw any light upon it. The question is whether you can ever get glycosuria as a result of muscular fatigue. I saw what was apparently a case of it some years ago in an individual who was apparently perfectly well, but developed transitory glycosuria as a result of extreme muscular fatigue. Whether it was a form of toxic glycosuria or not I do not know, for there is such a thing as toxic glycosuria, as everyone must admit.

Putting aside non-diabetic glycosuria, I pass to the diabetic to say a few words in the short time that remains. Surely one of the striking things about diabetic glycosuria is the very great diversity of the different cases of diabetes; and what we want at the present time is a thorough and more detailed classification of diabetes than we at present possess. The division of them into an alimentary group and a composite group is a very great step in advance, but one cannot help thinking that the composite group may perhaps have to be divided further. You may classify cases of diabetes in a great variety of ways, as, for instance, according to the age of the patient—and that is not such an artificial classification as at first it seems, because the malady does run a different course at different ages. Or you may classify them according to the state of the urine, as to whether sugar is the only abnormal substance present, or whether the urea is increased in quantity, or whether the total output of urine is increased. It would seem to one to be very necessary that we should, at any rate ultimately, have a classification based upon the morbid anatomy, or based upon the pathology of the disease. If we look at the matter from this point of view we must recognise that at the present time it is impossible to separate them clinically. Theoretically, there are at least five forms of diabetic glycosuria which would have to be recognised: first, the alimentary form, which rests on a secure basis from a clinical standpoint; it is a form which can not only be recognised, but can also be prognosed and successfully treated. The existence of that form is beyond question. Secondly, most observers would, I think, admit that there is a hepatic form of diabetes. The hepatic form may be simply dependent on the liver losing its normal function of stopping the sugar that comes to it, or the hepatic form may really be divisible into two—a slighter form in which the liver simply loses the function of stopping the sugar that comes to it, and another form in which the liver makes more sugar; it would seem that this is the more probable in the light of the fact that certain morbid lesions can be found in the liver in certain forms of diabetes, and that glycogen can be found in the living liver of the diabetic. At any rate I think that diabetes will be recognised as having sometimes a hepatic origin. Then, thirdly, there is the pancreatic type, which I think everyone will admit, and which, at any rate in some instances, it is possible to recognise clinically. In some cases of very acute diabetes associated with gangrenous and inflammatory lesions of the pancreas, the condition has been recognised and diagnosed during life. Fourthly, it would seem also that a renal type must be recognised, especially in the light of the experimental results obtained with phloridzin. With reference to this renal type, it is possible that the lesions that we sometimes find in the kidneys of the diabetic may have a new significance. It is well known that the kidneys are hypertrophied in diabetes, but not in all cases of diabetes. Some of the cases which have had the greatest excretion of urine may be found post mortem to have quite small, shrivelled kidneys. On the other hand, other cases may have greatly hypertrophied kidneys. I should have thought it was not proved that hypertrophy of the kidney associated with some forms of diabetes was simply due to the excretion of a large quantity of water; it is possible that it may have some other significance, and it may be that it is directly related to the glycosuria. And with reference to this, as other speakers have said, there may be a disappearance of the sugar in some forms of diabetes when a renal lesion supervenes, and that may also now receive a new explanation. The last possible origin of glycosuria is the least secure—namely, the hypothesis that there might be glycosuria from primary tissue changes. That, of course, was advanced long ago on theoretical grounds, and is based upon the breaking up of the proteid molecule into urea and into sugar. On theoretical grounds that is a view which one thinks might be defended. At the present time I think the four first which I have mentioned rest on a more secure basis, although one of course has to regret that they cannot be

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recognised and differentiated clinically. The nervous system has been left out in this consideration, because, although there is no doubt that the nervous system does play a very important part in the production of the malady, there is no sound experimental evidence, as far as I know, to the effect that it plays a direct part. And Bernard's experiments are more in favour of the production of glycosuria by irritative nervous lesions than they are in favour of the possibility that the nervous system can produce actual diabetes. Then with regard to diabetic glycosuria, other speakers have drawn attention to the fact that the sugar in diabetes disappears or may disappear after the onset of coma. It is a remarkable fact that the disappearance of sugar may precede the onset of coma by a number of days in exceptional instances. Sugar will disappear in diabetes apart from improvement and apart from coma under other circumstances, as, for instance, the onset of an intercurrent febrile illness, although this is not invariably the case. So there are a considerable number of conditions in which sugar will disappear from the urine in diabetes, and it will be interesting to have more accurate knowledge as to what is the true significance of this fact.

The Lesion in a Severe X-Ray Burn.—Dr. JAMES P. TUTTLE referred to a case in which he had been compelled to amputate the thigh because of the results of an X-ray burn. That was the only case in which a careful pathological and microscopical study of an X-ray burn had been made. After the amputation a very close study of the parts around this burn had been made by the late Dr. William Vissman. His conclusion was that the X-ray burn was the result of the production of an endarteritis and a periarteritis. The alteration in the capillary supply about the burn seemed to explain the failure to secure healing. This observation might explain the gangrene referred to by some. In his own case the endarteritis had extended three or four inches outside of the actual burn or slough, showing that the effect upon the arteries was not confined to the part which sloughed as a result of the burn.—*Medical Record*, April 4th, 1903.

WITH MR. JAMES BERRY

AT THE

MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

HERE is a woman who has a very obvious painful swelling on the inner side of the head of the first metatarsal bone. It is equally obvious that she has had over it a sore which has recently healed. Looking at the foot you will see that the first phalanx of the great toe is displaced to the outer side,—in other words, that the foot is in a condition of hallux valgus, with a bunion. The condition gives her considerable trouble, and she is therefore anxious to have some active treatment for it. Various forms of treatment have been tried without any benefit. Therefore I propose to do what is a very good operation for really bad hallux valgus, and that is to remove the head of the metatarsal bone. There is one point I want to draw attention to. If you are removing the head of the metatarsal bone for hallux valgus, it is essential that you should take away enough bone. A mistake which is sometimes made is to take off just the top of the bone; if that is done, the cut surfaces of the metatarsal bone and the phalanx are left close together, and either union takes place, with a stiff toe, or a considerable amount of new bone is thrown out. In the latter case the patient has still a painful swelling, and she is no better than she was before the operation. Therefore take away a considerable piece of bone, from an inch to an inch and a half, comprising the whole head and a portion of the shaft. In doing this operation take care also not to cut the long flexor tendon of the toe. Another method of treatment, which you may adopt for less severe cases of this deformity, is the use of an apparatus with a spring to keep the toe in a natural position. It is of no use, however, if the deformity has gone very far. In a case in which the deformity has gone as far as in this woman it is more satisfactory to operate.

The next case is one that I have not seen until a few minutes ago. It was sent up here by Dr. Belilios with a letter giving the following particulars:—The patient is 58 years of age. Over three months ago he was in a hospital in London for irreducible left inguinal hernia, for which an opera-

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tion was done. At that time nothing abnormal was noticed about the abdomen except the hernia. When coming round from the anæsthetic he appears to have been violent and to have pulled all his dressings off. One result of that was that the wound suppurated, and an abscess formed which had to be opened and drained. When he returned to his home there was a healthy granulating wound. Nearly three months ago, about ten days after his discharge from the hospital, while he was straining at stool, he suddenly felt an intense colicky pain in the upper part of the abdomen. On palpation about an hour afterwards a swelling of the shape and size of a pear was felt in the upper part of the abdomen, about an inch below the left costal margin. The swelling broadened out below and extended to a point one inch and a half below and to the right of the umbilicus, where it is now easily felt. There was slight jaundice at that time, with high-coloured urine. He had intermittent fever, a temperature of 101° or more in the evening, subnormal in the morning, and he sweated a good deal. The pain was so bad that he had to have morphia. He had intense pruritus, evidently due to the jaundice. The swelling is reported to have diminished in size. His general symptoms improved, but the jaundice continued. In seven days the swelling was of the size of an orange, in the middle line above the umbilicus. There was again fever and increased jaundice. The tumour, by the report, appears to have varied in size, and although, on the whole, since the operation he had lost flesh, yet in the last month he has not got any thinner. There was nothing at all the matter with him, as far as he knows, except the hernia, until the date of his operation. We see the scars of the hernia operation. The wound is healed, and there appears to be nothing wrong with it at present. The patient's general health appears to be fairly good. The looseness of his skin, however, suggests rather that he has been losing flesh. The abdomen also is rather flabby, and suggests that it has been larger than it is now.

Directly I lay my hands upon the abdomen I find there is, as the notes told you, a considerable swelling in the centre of the abdomen. It extends two and a half inches on either side of the middle line, and below the umbilicus for one inch, and passes upwards for some three inches, to within one inch of the ensiform cartilage. This swelling is exceedingly hard, apparently not very tender. It is tolerably smooth on the surface, and slightly movable from side to side. I gather that this trouble came on within a week or so from the date he was discharged from the hospital. At that time he had a suppurating wound, and that seems to me an important point to remember. It suggests a cause for this lump. There are, I think, two diagnoses which we must consider. One is that this lump is directly connected with the suppurating wound. If so, it is some sort of inflammatory swelling. The other is that he has an independent lump in the abdomen, which has nothing to do with the hernia, or the operation, or the wound. In that case the swelling may possibly be of a malignant nature. I think there can be no doubt at present that it is solid. The history of an acute attack, the severe pain, the high temperature which he had for some time, and the sweating, all point to some inflammatory trouble. The fact that the swelling was at first very much larger, and subsided distinctly in the first three or four weeks, also points to an inflammatory condition; so does the smoothness of the surface. I am inclined to say that this is more likely to be inflammatory than malignant. But I am well aware that sometimes a malignant tumour connected with the hollow viscera may present a very large mass of inflammatory tissue around it, and it is possible that there is some malignant growth behind what we can see and feel. Some months ago I showed you a young woman who came under my care with a swelling very similar to that which is now before us, but rather more nodular. I thought it was probably a malignant tumour of the omentum or mesentery. I opened the abdomen and found a firm, hard, nodular growth in the mesentery, firmly incorporated with the small intestines, and apparently an obvious sarcomatous tumour. I closed up the wound at once and gave a very bad prognosis. From the time of the operation the tumour steadily decreased in size, and in three months there was no trace of it. I saw this patient again recently, and found that she had remained perfectly well.

That is one example of a class of case which is very well known, and of which examples are not very uncommon. I dare say most of those present could tell of a case of a similar kind, in which an

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abdominal tumour apparently of malignant nature disappeared entirely after an exploratory abdominal section. I have no doubt that the woman whose case I have related had a mass of tuberculous or chronically inflamed glands in the mesentery. I do not think anyone could say positively, without watching this case a little longer, whether it is inflammatory or whether there is something more serious behind it. But I think it is more likely to be purely inflammatory than anything else.

[The subsequent history of this case was that the tumour soon disappeared entirely.]

This old man I saw yesterday for the first time. He is a tailor, sixty-five years of age. He came to the hospital because he has suffered during the last ten weeks from diarrhœa. Up to that time he was perfectly well. Then, rather suddenly, diarrhœa began, and this lasted more or less continuously ever since He tells me that sometimes for two or three days he has no diarrhœa, but that is the longest interval of freedom from it. There is nothing peculiar about the diarrhœa, and he assured me that he never passes blood with his motions. The bowels are opened four or five times a day. For some months he has been feeling ill, and he thinks he must have lost two or three stones in weight, although he cannot tell us exactly what his weight was. He has not been aware of any lump in the abdomen, and it was not until the swelling was pointed out to him that he knew he had the lump which we can now feel. As in the last case, there is not much to be seen about the abdomen. Here also the skin hangs rather loosely, and that confirms the opinion which has been expressed that he has lost weight. His abdomen is somewhat flabby, and rather suggestive of an abdomen which has been at some time rather larger than it is now. I think you can all see that there is a swelling to the left of the middle line of the abdomen. When the patient sits up the swelling disappears; that shows that it is behind the anterior abdominal wall. When I put my hand upon it I feel a very obvious firm swelling, which extends one inch to the right side of the middle line. Below, it comes an inch or two below the level of the left anterior superior spine. Above, it extends to within one and a half inch of the ribs. Laterally it extends practically to the lateral wall of the abdomen. It does not go very distinctly into the loin; when I put my hand below the lower ribs I can feel the tumour, but not very plainly. Certainly it does not bulge at the loin. On the right side and in all other parts of the abdomen I find nothing abnormal. The liver certainly is not large, and there is no evidence of any disease in it. The patient has no urinary symptoms, and is not aware that he has ever had any. Examination of the rectum reveals nothing abnormal. He is not sick. So we have no symptoms at all pointing to a diagnosis, except the diarrhœa extending over three months, and the gradual loss of flesh during the same period and longer. We know that he has had this swelling for some days, and probably for several months, as he was not aware of it until it was pointed out to him. The swelling is exceedingly hard, firm, and tolerably smooth. At one point lower down there is a small, hard lump, apparently a little outgrowth from the swelling, about the size of a cherry. And at the upper part there is another lump, which apparently is not part of the main swelling. The spleen is in its normal situation; at any rate, there is a patch of dulness at the usual place for it. We must now consider another point, namely, the relation of the tumour to the colon. The tumour in front is dull. Round in the loin it is resonant. The colon I cannot definitely feel, although I have felt carefully over the front of this swelling to see if I could detect it. If I could feel a definite ridge running vertically over the front of the swelling I should say it was probably the colon, and that would point to the tumour being a renal one. I have tried to determine the situation of the colon in another way. I have had the colon inflated with air, and the distended colon was then found to lie on the outer side of the tumour, somewhat in front of it.

Now about the diagnosis. Here is a lump, and we want to know two things about it. First of all, where is it? In what anatomical structure is it situated? Secondly, what is the nature of the lump? I think both these questions are very difficult to answer. First of all as to its situation. It is an oval, firm lump, which is clearly behind the anterior abdominal wall. Clearly it is not in the posterior abdominal wall, because it moves from side to side. It is therefore either loosely embedded in the retro-peritoneal tissue, or it is in the abdomen proper. Its situation suggests that it is either a swelling of the kidney or of the

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intestine, or that it is a swelling proceeding from the mesentery or the retro-peritoneal tissue. With regard to the kidney, there is an absence of any urinary symptoms, but that does not prove that it is not kidney, because the kidney may be very much enlarged and yet may give rise to no symptom whatever as regards the urine. If the ureter is blocked, and if the trouble involves the substance of the kidney and not the pelvis, there is no reason why there should be any urinary symptoms at all. The situation, however, is not quite characteristic of kidney. The swelling does not bulge the loin in the way most large kidney tumours do. But we must remember that a renal tumour is not necessarily situated very far back, and sometimes shows a tendency to come forward on to the front of the spine. I have seen a kidney lying almost directly in front of the spine. Some times kidneys are congenitally situated in some abnormal position. Some of us have seen kidneys situated on the brim of the pelvis. So the fact that this tumour does not occupy the usual situation of the kidney does not prove that the case is not of renal nature. With regard to the colon, if it be true that this really is the colon lying outside and somewhat in front of the swelling, that would tend to show that this is a kidney, and that it is especially the inner part of the kidney which is enlarged.

Let us turn now to the question of it being something in the intestine, or in the mesentery, or in the omentum. As a rule a tumour of those parts is very much more movable than this; the mesentery or omentum will move much more freely than this. It may be that this tumour was more movable before, and that it has now become more fixed. Apparently it has not become fixed by inflammation, for there is nothing pointing to acute inflammation here, either in the history or in the present appearance of it.

Malignant growths of the intestine are usually comparatively small. They are seldom larger than a Tangerine orange, and may not be larger than a walnut. This swelling is far larger than that. Sometimes one meets with large abdominal swellings in elderly people which are composed partly of growth, but mainly of inflammatory tissue around the growth, or possibly of fæcal matter above a malignant obstruction. I ask myself whether this can be merely a fæcal accumulation; and I say no, because when I try to get pitting I fail absolutely. But there is one kind of tumour of a carcinomatous nature in the intestine which sometimes attains a large size, and that is colloid carcinoma. But a colloid carcinoma is not as firm as this is, and would be much more irregular in shape. I do not think this tumour can be colloid. I do not think we can exclude the possibility of this tumour being a sarcoma of the mesentery, but it is a rare disease, and there is not sufficient evidence in this case to enable one to diagnose it with much certainty.

With regard to retro-peritoneal tumour, it is always dangerous to be unduly influenced by a case which one has seen before of an apparently similar nature. Some two years ago I saw a man of about this age who had a tumour of similar size and shape, in a similar situation, and with a similar history; that is to say, there was an indistinct history of a lump and of diarrhœa, and I was in considerable doubt at first whether it was not a case of carcinoma of the rectum with surrounding inflammation. But that proved to be a large retro-peritoneal sarcoma, occupying a part of the pelvis. I hesitate very much to say this is a case of the same kind, especially as that tumour was more in the pelvis, and this is higher up. I do not feel prepared to give you a more definite opinion than I have. I do not see how it is possible to get nearer to a diagnosis without making pure guesses. It is uncertain whether it is a kidney tumour or a swelling outside that organ. If it is a kidney swelling I think it is most likely to be an old pyonephrosis or hydronephrosis, possibly a malignant tumour. Blocking of the ureter would account for the absence of urinary symptoms. I should be more inclined to diagnose retro-peritoneal sarcoma if I did not know it was a very rare disease, and whenever possible prefer to diagnose a common condition rather than a rare one. The diarrhœa is the one prominent symptom which points to some intestinal trouble, and it is difficult to explain it on the assumption that it is some tumour entirely unconnected with the intestine.

[The subsequent history of the case was that the patient gradually got more and more weak; the diarrhœa continued, and in five weeks he died. No fresh symptoms occurred. At the post-mortem it was found that the tumour consisted of coils of sigmoid flexure and upper part of the rectum matted together by chronic inflammation set up by a carcinomatous tumour of small size which had perforated the intestine in several places. The left kidney lay in contact with the tumour, but was not involved in it.]

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A CLINICAL DEMONSTRATION

At the Hospital for Sick Children, Great Ormond Street, W.C.

By F. G. PENROSE, M.D., F.R.C.P.,

Physician to the Hospital.

THERE happen to be now in the ward several cases, each offering one or two points of interest, to which I wish to draw your attention.

I want you to remember that in nearly all these cases there is a certain element of doubt. You know that it is always said that the proper method of teaching medicine is to be dogmatic, to say that the thing is so and leave it there. That is perhaps a good plan when men are first beginning their training, but to attempt to dogmatise to you, who are a great deal further on than just beginning, is unwise. If one does so there is a certain room in almost every hospital which has a habit of revealing that one's dogmatic words are not in accordance with facts.

I propose to point out my own ideas in these cases, but I shall put forward others so that you can come to your own conclusions.

A CASE OF PERICARDITIS WITH DISTENDED ABDOMEN.

This child will not detain us long. She is a little girl of 3½ years old who comes into the hospital with very well-marked pericarditis. There are well-marked intra-cardial murmurs, and there has been a friction rub which we have followed since she has been in. There remain some little odd clicks which should make you suspect, when you hear them now for the first time, pericarditis.

What I wish to point out is the distended abdomen in this case. I have occasionally been very much struck by the fact that in chest cases when the abdomen begins to become distended it is a bad sign. It is generally ascribed to muscular weakness. This child was very ill when she came in with the pericarditis. It is a little clinical point

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that is sometimes of very great value indeed with regard to prognosis, this gradual distending out of the abdomen with intestinal gases in cases of serious disease. I do not think it quite holds good in this case, having watched the child for a month. This child is gradually improving in health, she is distinctly better than when she came in, yet the abdominal distension has only slightly decreased. Of recent years in this hospital a good deal of attention has been paid by Dr. Still to inflammatory conditions in connection with the pericardium in cases of tuberculosis. He collected a small number of cases of well-marked pulmonary tuberculosis in which there was a certain amount of pericardial inflammatory change to be seen at the base of the heart, and especially in the pocket behind, where the pericardium goes back and folds over.

That I took as the next point to consider in this case. Is this a case of tuberculous peritonitis giving us the distension, and can there be also tubercular pericarditis?

All I can say is that that point is still in abeyance. I do not see enough evidence to show that it probably is so. We can find no definite evidence of tuberculous lesions in the abdominal or thoracic viscera.

You will see that I am puzzled to give you the real significance of this distended abdomen. I believe the heart lesions, as in so many of the children here, are rheumatic in origin. I have put before you two theories in regard to the abdominal distension. I am not at all certain as to the cause, and that is why I gave you the little premonitory exhortation as to dogmatism. There is no evidence of enlargement of the liver, probably the commonest condition found in heart disease where there is abdominal distension; nor is there any evidence of rickets, which, as you know, is often accompanied by a *pot belly*.

A Case of Congenital Heart Disease.

This child is 11 months old; described as a strong baby with no particular illness except that it has had a cough for some six weeks and has been getting thinner. After it came into the hospital we found how abnormal its internal organs were. The child, you will observe, is not blue (there is some account of its having had attacks in which it became blue). There is no clubbing. You see the fairly healthy colour of the child. If you look you will see that the apex-beat is here in the sixth or seventh space, one inch outside the left nipple line, a position for it that is quite uncommon in infancy. In this child it is due to the large size of the left ventricle.

The cardiac dulness to the right of the sternum is hardly outside its normal position. You will recognise the peculiar, loud, almost continuous murmur, heard all over the precordial area, and conducted specially round here to the upper part of the left side. It is impossible to say whether it is systolic or diastolic. It is a loud rumble running pretty well through the cardiac cycle.

The next point to bring before you is that we may to a certain extent make a guess—and please remember it is nothing more than a guess—of the kind of cardiac lesion that is present here. At various times by various clinicians a great deal of energy has been expended in trying to come to a conclusion as to whether, in congenital heart disease, we have any indication of the kind of lesion present. I think the only lesion that is fairly definite is where there is a large patent foramen ovale with stenosis of the valves of the pulmonary artery. In that case you have a murmur conducted specially up to the apex of the chest, and a tendency for a greatly increased pulsation at the root of the neck. There is one such case on record in which a young woman lived to over thirty years of age with this condition, and this big thumping pulsation was taken to be an aneurysm, and one of her carotids was tied. It was a case of Mr. Timothy Holmes, and was published in the Clinical Society's 'Transactions' (vol. xxi, p. 146).

I have seen a little child here with the same lesion and with the same symptoms.

The most common malformation is a want of proper formation of the intra-ventricular septum. In almost all these cases the right ventricle, which, as you know, is during fœtal life larger than the left, remains large. I have therefore ventured to put that aside.

And so I do not consider this case to be one of failure of the formation of the intra-ventricular septum, nor do I think that it is one of patent foramen ovale with pulmonary stenosis.

One is sure that almost all the work is being done by the left ventricle, and there is probably a direct communication between the left ventricle

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and the pulmonary vascular cycle; and I suspect we have some communication between the aorta and the lungs in some way or other.

There is one practical point about all these cases. Very severe heart disease may be present, and there may be no blueness and no clubbing. Some of these children live to a fair age, but, however well they seem to get, remember that the prognosis is always unfavourable. These cases of severe congenital heart disease nearly always die young, usually of consumption. The circulation does not seem to be properly carried on through the lungs, and apparently the resisting power against the tubercle bacilli is weakened.

A Case in which the Left Pleural Cavity was Opened.

This case is one of importance. The child is six and a half years old. There was a cough last Christmas. She was admitted on March 31st. In the third week of January she caught cold and had bronchitis. In the first week in March, at Romford Hospital, the chest was explored twice. First clear fluid, and the second time thick fluid, was obtained from the left side of the chest.

When she was admitted the heart was displaced two fingers' breadth to the right. The left side was dull all over. The case seemed to be urgent. We had her history; and Mr. Fairbank, the resident medical officer, considered the case critical, and she was put under an anæsthetic and a portion of a rib was removed. When he examined the chest there was no fluid present in the pleural cavity; the lung was adherent. He opened into the lung and came across a lot of cheesy-looking stuff; I do not mean definite caseous material, but the sort of stuff you see in the periphery of lymphatic gland that has been at one time caseous and has a little calcareous nodule in it from an old tubercular abscess. The whole of the left lower lobe was apparently composed of this white friable stuff.

I saw the case later. The displacement of the heart seemed to point so strongly to empyema that a needle was inserted, which gave us stuff that we took to be pus.

A second operation was done and a second piece of rib was removed, and again no fluid was found. Mr. Fairbank found adhesions around the old wound, but the pleura was otherwise all free and clear. He went right into the lung, and again got out this same friable stuff. It was subjected to microscopic examination. That was negative. The only suggestion was that it might have been a sarcoma. The stuff removed was not in properly organised tissue, but consisted simply of various round-cells.

The history shows, I think, that there must have been a pleurisy to begin with; the clear fluid of which we hear about at the Romford Hospital must have been a pleural effusion. (There is no suggestion of the pericardium having been tapped.)

The next time when pus-like material was drawn the needle apparently went into the lung.

But here was the heart displaced to the right, so one might reasonably infer the presence of fluid, this being the usual, though not invariable cause, of the heart's being displaced to the opposite side. I have seen a sarcoma of the right lung push a heart into the middle of the left axilla. Tubercular conditions may draw the heart to the affected side.

We failed to find fluid in the pleura. Anyone looking at the child would be inclined to say there is no great difficulty in making a diagnosis; tubercle is stamped in the child's face. She has long eyelashes, thin pearly conjunctivæ, long limbs—all signs of a child that comes of tuberculous stock; and she has the bright-coloured cheeks which in ordinary parlance would be taken as a hectic flush. But very few of us have seen a child like that with a hectic flush due to tubercle having the whole of one lung solid as far as you can reach it, with a temperature chart such as I show you. (The temperature was practically normal.)

Empyema we know it is not. Seeing is believing, and seeing and feeling is more convincing. She has some pus-like stuff in the chest, but it is from the lung. The openings that were made are granulating up, and there is a certain amount of suppuration in the lung, which is to a certain extent pulling her down, but I hope to get her away into a better air.

We may exclude empyema. We may be very doubtful about tubercle. What I have said about tubercle holds equally well about sarcoma. But a child with a lung full of sarcoma cells, who has been operated on, would not be as happy and as well as she is. A child with sarcoma of the lung that is suppurating is always extremely ill. This child does not look that. I am driven back to the conclusion that the most probable thing is that at

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the original attack she had pleuro-pneumonia, that the pleurisy was absorbed, and that she now has a very chronic pneumonic condition of that left lobe, which, perhaps owing to poor health and being in London, is not healing up as it should. (I have heard that the child has recently died, and that no examination was made.)

A Case of Chorea with Tachycardia.

This child is nine years old. She came into this hospital on March 12th. For the last four months she has been noticed to drop things; she cannot feed herself, and sometimes falls down. She is said to have been frightened by a boy, who threw a potato at her head and knocked her down.

There is no history of rheumatism, nor of any rash, but she has sometimes had a sore throat. The mother was possibly rheumatic. The child, as you see, is a jolly-looking, well-nourished, full-sized girl for her age of nine years; at the present time her chorea has practically stopped.

I can find little in the history suggestive of rheumatism beyond the sore throats. She has complained of pain in the left leg. She has chorea, and her palms and soles are a little clammy. She has no cardiac murmur, no real joint-pains, and no enlarged glands. The condition of her heart is what is so interesting. When you examine her, listen to it, and you will find that it is going at an extraordinary rate.

When she first came in its beats were difficult to count—somewhere about 200. Whenever she is awake it is running somewhere about 200,—that is to say, as high as you can count. (To the unaided finger it is difficult to count beyond that figure; I cannot myself be sure of anything much over 160; some people can, however, count up to 200.) She has lately been getting better from the chorea, and her pulse-rate has gradually come down, so that sometimes it does not run up above 108. During sleep it has always been less frequent—from 100 to 120. We kept her in bed and hardly allowed her to move, but this apparently made hardly any difference. She is now up and about without her cardiac rhythm being altered at all.

Valvular disease and pericarditis with the choreas after rheumatism are common; but I never remember seeing before a case of tachycardia coming on in a case like this where the child is well, where there is no sign of cardiac enlargement, or of disease of the valves, muscle, or pericardium. There is no murmur; the heart is of normal size. She may have an adherent pericardium that is impossible to diagnose, but there has been no history of any cardiac trouble. When pericarditis has been present there is generally some history of the illness.

I have seen transitory palpitation in cases of rheumatism, but a continuous tachycardia like this I have not seen before. It does not seem to do her any harm; the child is well and bright.

I believe that it is a rheumatic case, though the other evidences of rheumatism are not very complete.

She is one of the old-fashioned sort of chorea that start with a fright. If you look through the notes of fifteen to twenty-five years ago, you will find that it was one of the things we laid stress on. A history of fright as the immediate cause was often put down. If you read the histories taken during the last five years, you will find the question is incidentally referred to, but not so much notice is taken of it as it was before. We have gradually come round to the belief that the all-important underlying factor is the rheumatic poison.

There is one other point; my colleague, Dr. Hutchison, has called attention to it. He lays some stress on the shape of the two upper central incisors, if they are big and square, and particularly if they are separated as well. He is inclined to think that it is of some value as denoting children that come of a rheumatic stock. We want more information about this point from other places, and I hope all of you, when you come across rheumatism, will note this point.

A Case of Tubercular Peritonitis.

Here is a little boy 10½ years old who was quite well till two months ago, when he began to waste. There has been no vomiting. He has been feeling tired, lying about a good deal, not ready to play, and rather complains of feeling cold and generally down. That is about all that I need say.

What I want to draw your attention to is that he has the unmistakably podgy abdomen of tubercular peritonitis. He has the skin that these patients so often get; he is not yet so wasted as they often are.

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Professor Koch rather startled us over here last year by the opinion that human beings did not get directly infected along their alimentary tract by tubercle from articles of diet, and especially from milk. Tubercle bacilli were an infection from the air; and when the alimentary tract got infected it was from swallowing sputum and from air-carried organisms, and not from contaminated food and drink. I think we ought to suspend our judgment on this point until we know more, but it is a question of the utmost importance.

The impression before Professor Koch's paper was that tubercle bacilli might infect the body in all sorts of different ways, although it was well understood that tubercular-laden dust was the most potent cause.

This boy was well till two months ago. We can find no evidence of tubercle in his upper passages at all—nothing in his throat, nothing in his chest. He gets a very rapid dissemination of tubercle in about two months in his abdomen; and I think this is a case which we should keep in our minds as to whether this may not be a case of infection from food products in some way.

A Case of Probably Mental Deficiency in an Infant.

Here is a baby eighteen months old. It was a full term baby, a fine child at birth, but wasted from the third day. Principal food: thin beef tea and various foods, etc. It has not yet got a tooth. It is not rachitic. It is very backward. I want you to take special notice of the look of its eyes and face. It suggests to me very strongly that it is not quite all there. It is impossible to put it into any recognised class of imbecility, but my impression is that the child is to a certain extent mentally deficient. She is extraordinarily passionate. At the present moment she is placid enough, but at times she is uncontrollably passionate, even for the age of eighteen months.

Little children that are "all there" have plenty of passion, but they are not quite so uncontrollable. We have considered whether there were any signs of any congenital disease. There are no signs of congenital syphilis; the child is not rickety; the only point is that the anterior fontanelle is still a little more widely open than usual at her age.

It is from its puny condition, from its backwardness, from its extraordinary fits of passion, and from a certain look that the child gets into its face, that gives the impression of an incomplete mental state. It is one of the doubtful cases. It is well worth watching, and I shall endeavour to keep my eye on this child to see how it develops.

Relations between Epilepsy and Migraine.—William Strohmayer ('Münch. med. Wochenschrift,' March 10th, 1903) concludes from a study of the material available in Binswanger's clinic in Jena that migraine and epilepsy often co-exist in the same patient. Usually migraine appears first, to be followed sooner or later by epilepsy. No case has, however, been observed by the author in which one of these diseases was transformed into the other, or in which one was substituted for the other. When such was apparently the case, it is evident that the migraine was only a symptom of the epileptic condition, or that the epilepsy developed as an independent disease, in addition to the migraine. In atypical cases it was difficult to decide whether the epilepsy or the migraine was the primary condition.

Banti's Disease and Anæmia Splenica.—F. Marchand communicates that splenomegalia is the cause of the cirrhotic changes which develop in the liver in the latter stages of Banti's disease, but that such cirrhotic changes are due to other causes, as, for example, congenital syphilis, or alcohol. Two cases are reported, one of Banti's disease and the other of anæmia splenica. In the former, the liver cirrhosis either preceded or arose simultaneous with the splenic condition. The case was operated upon for the ascites, and thereafter the cirrhosis became non-progressive, and at death autopsy showed that the liver condition was not active. In the second case, the patient became tuberculous and died. At autopsy no cirrhotic lesions were found in the liver, although the patient had for some years suffered from splenomegalia.—*Medical Record*, April 11th, 1903.

Writing on the treatment of chronic articular rheumatism, Dr. Burney Yeo says that *aspirin* has been favourably reported on. It does not disorder the stomach nor cause circulatory depression.

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REST IN THE TREATMENT OF HEART DISEASE.

By CHARLES W. CHAPMAN, M.D., M.R.C.P.,
Physician to the Hospital for Diseases of the Heart, Soho Square, W.; late Physician to the Farringdon General Dispensary, E.C.

It was noted by one of the reviewers of my book on 'Heart Disease in Childhood and Youth' that I had not given sufficient prominence to the value of rest as a therapeutic measure. I had perhaps taken it too much for granted that its importance was already fully recognised. But, considering the insistence with which opposite mode or modes of treatment have been advocated of late years, it is perhaps well to again put forward the unique claims of rest.

The mistake has been made in fostering in the lay mind the idea that all methods of treatment have been superseded by baths and exercises, and that a certain German health resort is a veritable Mecca for cardiac patients of all kinds, and at all stages of the disease; whereas a large proportion of such would reap more lasting benefit by treatment based on the recognition of the particular requirements and varying condition of each case. The truth is, no one plan of treatment is of universal application; and I trust I shall be able to show that, without disparaging other therapeutic measures, rest has its own special place in the successful treatment of many forms of heart disease.

The importance of rest in the treatment of both acute and chronic cardiac affections has long been known; it may, however, be profitable to consider in detail the indications for rest and the rationale of its application.

1. Pain is a common symptom in all forms of heart disease, and it varies in intensity and importance in different cases. Now assuming that the numerous extra cardiac causes for pain in the heart or its neighbourhood have been eliminated, such as pleurisy, intercostal rheumatism, and neuralgia, indigestion, etc., the existence of pain, especially when provoked by exertion, is an indication that rest is necessary for successful treatment. It is noteworthy that external applications of a sedative nature are not of much value for true cardiac pain. Belladonna is frequently used for this purpose, but not with very satisfactory results. Mr. Hilton in his classical lectures on "Rest and Pain," after showing the relief that can be obtained in some chest affections . . . . "through the medium of anæsthetics applied to the cutaneous nerves associated with the cardiac nerves of the pleura costalis," continues thus: "But I admit we have very little opportunity of acting directly upon the heart or pericardium through the external or cutaneous nerves associated with the cardiac nerves, so as to induce physiological rest in that organ by the external application of anæsthetics. You will be impressed with this conclusion when I remind you that there are but a few filaments of the upper intercostal spinal nerves which join the cardiac plexus of the sympathetic nerve within the chest; and that these same intercostal nerves distribute only a small number of filaments to the skin of the chest and back. It must not, however, be overlooked that the cervico-spinal nerves communicate with the cardiac nerves derived from the sympathetic ganglia in the neck. These are apparently the only nerve-trunks extending from the surface of the body to the heart which would permit of direct anæsthetic influence being propagated from the skin to the heart."

As pain is one of the danger signals in heart disease which points to the necessity for rest, not only for its relief, but for the functional ability of the heart itself, it is well so kind but cruel a monitor should not be too easily silenced.

Probably the chief cause of pain in chronic cardiac disease is distension of one or more of the chambers of the heart. A. S—, æt. 14, came under my care in June, 1901. He had rheumatic fever severely at 7 and 13½ years, but he had kept in good health up to December last. He complained of palpitation, dyspnœa, especially on exerting himself, and an occasional sharp pain at the heart. There was considerable bulging at the præcordial region, heart's action was forcible, and the apex-beat was at the sixth space. There were double aortic and mitral systolic murmurs. The patient's own words were:—"When the palpitation stops, severe pain at the heart comes on, which is relieved when the heart beats well again." This symptom commonly occurs in valvular disease, when the heart is called to do work beyond its power by exertion on the part of the patient. The compensation may be good enough to enable the heart to carry on the circulation while the body

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is at rest, but the balance may be so delicate that a little strain will embarrass its action and lead to an increase of intra-cardiac tension which the weakened muscle is unable to cope with, and pain is one of the results. As the case advances on the downward grade pain may follow the least voluntary movement, and the oft-repeated demand for rest has to be obeyed. Now as the chief aim of the physician is to prevent this breakdown, he orders more or less complete rest when pain and other signs of commencing cardiac failure present themselves, and he seeks by treatment to restore the functional capability of the heart. One of the most valuable of these means is rest.

Another indication for rest is the occurrence of dyspnœa, which, like pain, is a sign of want of due compensation. Dyspnœa is an indication that the right heart is involved; hence its frequency in the later stages of most heart cases. If the failure be primarily of the left ventricle, its cavity, not being properly emptied, is unable to receive the full complement of blood; the blood-pressure in the pulmonic circuit is raised, and a strain thrown upon the right side of the heart. In these cases active measures, such as the application of leeches, or even venesection, will often give greater relief than stimulants by relieving the strain on the right side of the heart, and so enabling the chambers to contract upon their contents.

Inflammation of a serous membrane is nearly always accompanied by pain, and nature endeavours to mitigate its severity by limiting the movement of the inflamed part or parts. In pleurisy the patient involuntarily fixes the chest on the affected side; in peritonitis the abdominal muscles become rigid; and in arthritis the muscles around the joint, by rigid contraction, ensure comparative rest. The heart, however, must work under all circumstances.

In acute affections of the heart, pain, though generally severe, may be not a very marked symptom, yet rest is essential. The aching, cutting, or boring pain common to acute pericarditis is so urgent that it calls loudly for relief, and opium, in such cases, not only makes the pain endurable, but calms the excitement of the organ and thus gives it comparative rest.

In heart failure rest is an indispensable element in successful treatment, and this is especially true in the early stage of the affection. If the dilatation is to be remedied it must be by sparing the heart all avoidable work, and by improving the condition of the myocardium. It is distressing to see children who have had to continue drill when, if the effect of such exertion had been noticed and rightly interpreted, much damage to the heart might have been avoided. Errand boys often present themselves for treatment when they can scarcely move without dyspnœa quickly supervening. Examination in such cases generally reveals extreme dilatation of the heart as well as valvular lesions, the dulness extending an inch or more beyond the right costo-sternal margin, and on the left to the anterior or mid-axillary line.

Rest as a therapeutic agent must be considered in two aspects: (1) functional to the heart itself; (2) bodily and mental rest. Under the first category would come the use of opium, already referred to; and bleeding, by giving relief to distension at the right side of the heart, especially when there is engorgement of the lungs evidenced by cyanosis. Dr. Lees has emphasized the importance of recognising dilatation of the right auricle. His remarks on this subject in his presidential address to the Harveian Society may well be quoted: "Very few percussors pay the least attention to this structure [right auricle], and many seem to be unaware of the fact that dulness due to it may always be detected, even in the normal heart, in the fourth right intercostal space. The third space ought to be resonant quite up to the sternum, and in the fifth space the hepatic dulness alters the note, but in the fourth space the dulness of the right auricle is present for about one finger's breadth in the adult and rather less in the child. When the auricle is dilated it may extend to two fingers' breadth in the fourth space, and from one and a half to two fingers' breadth in the third, and may even be detected in the second. The accurate determination of the size of the right auricle is a matter of the greatest importance, and often indicates at once the necessity for leeches to relieve distension." No one who has witnessed the improvement in cases of right auricular distension by the abstraction of blood can doubt the value of the procedure. The baneful effects of alcoholic stimulation in such cases I have written on elsewhere. *

* "The Rôle of Alcohol in the Treatment of Heart Disease," 'The Lancet,' 1895.

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The relief witnessed in cases of hypertrophied heart associated with high arterial tension by a dose of calomel is, in a great measure, due to the "opening up" of the capillary circulation by the removal from the blood of the results of faulty metabolism, thus diminishing the strain upon the left heart. In the cardiac dilatation met with in cases of anæmia, rest, more or less complete, is essential. How many a work-girl and domestic servant would be saved from becoming a permanent heart cripple if rest had been taken as soon as the heart showed signs of dilatation.

Cases of aortic regurgitation not infrequently recover much of their lost compensation over and over again if rest is resorted to as soon as there are signs of fresh yielding of the left ventricle. When the patient assumes the recumbent or semi-recumbent position the weakened left ventricle is but little taxed in sending the blood on so gentle an incline, and comparative rest is obtained, while at the same time the myocardium is strengthened by strychnine and other means. When the later stages of valvular disease have arrived, and the lungs are gorged or œdematous, the liver enlarged, and possibly ascites present, the patient has to be propped up with pillows, and ultimately is compelled to spend his time in the chair. Under these circumstances it is the greatest cruelty to insist on the patient keeping his bed. With laboured breathing it is obvious that the ribs and diaphragm can act better in the sitting posture. Moreover when there is more or less general œdema the fluid can gravitate to the legs and be drawn therefrom much more satisfactorily when the patient takes to the chair. These patients are often unable to sleep, and the insomnia has a wearing effect upon the nervous system which aggravates the struggles of the labouring and faltering heart. The beneficial effect of a dose of morphine combined with strychnine is most marked at this juncture by giving the required rest. The altered expression in the faces of such patients after a few hours' sleep is evidence of the value of the sedative.

After a cardiac patient has had rest and benefited by it, the question arises whether the heart has sufficiently recovered to stand the strain of passive exercises. By noting carefully the position and strength of the apex-beat and limits of cardiac dulness, then watching the effect of a few resisted exercises, an approximate estimate may be formed of the capabilities of the cardiac muscle. If the action of the heart becomes stronger and the pulse becomes slower and generally improved under stimulation, the exercises can be persevered with, while if the action tends to increased embarrassment of the circulation further rest must be enjoined, and the nutrition of the general muscular system kept up by judicious massage for a further period. Cycling on the level, with more or less frequent intervals of rest, is one of the best forms of exercise in suitable cases.

When the patient's circulatory system improves with muscular exertion, adequate intervals of rest must be enjoined even when compensation has been restored. In other words, the physician must in each case "feel his way." In aortic cases pallor and faintness are the signs that the heart is bearing the strain badly; while in mitral disease dyspnœa and cyanosis are the danger signals.

Young patients with aortic reflux whose compensation has given way under extreme muscular stress, but who retain a fairly good second sound at the base, recover wonderfully if rest is early resorted to and sufficiently long maintained. It is more surprising still how the youthful heart will recover its working capability over and over again under rest and treatment. Mitral cases do not as a rule respond so readily and completely to the same measures. When, however, we are dealing with heart failure in patients who have arrived at the degenerative period of life, or those who have anticipated this stage by alcoholic excess, the beneficial effects of rest are not so well marked; it is, however, more rather than less needful on this account. To send such a patient on a long journey for the purpose of treatment without being assured that he is in a fit state for travelling, and that it is probable he will derive benefit from the visit, is to run a risk that should not have medical sanction. Cases of very chronic Bright's disease where a cloud of albumen has been noticed for years, yet the patient has gone about his work without let or hindrance, at times suddenly manifest severe cardiac symptoms. Such a case is at present under my care, the patient being a professional man forty-five years of age. He had had a trace of albumen in his urine for ten years without any inconvenience arising from it. Suddenly last December, after taking a cup of black coffee, he was attacked with severe angina, which, from the character and

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area of the pain, was supposed to indicate aneurysm of the aorta. The heart was found to be hypertrophied, but devoid of valvular lesion; thorough investigation gave no evidence of aneurysm, leaving the subjective signs alone to suggest that condition. Moreover the urine was of the same character as it had been for years. The patient would not consent to forego his engagements, and he elected to try the effect of medicinal and dietetic treatment. He got on fairly well during the day, but the nights were distressing, so that he was compelled to rest. The symptoms were speedily removed by rest and medicinal treatment, and the patient in a short time returned to his normal state of health.

Another form of rest so valuable when it can be obtained is that of the mind. The necessity of mental rest should always be borne in mind in treating severe cardiac cases. In the instance just related the comparative importance of physical and mental rest had to be considered. Here was a man of an anxious and excitable temperament who had important engagements on hand, to whom rest would be impossible unless his mind was easy. I therefore concluded he had better get his business done with as little exertion as possible, and then lie up.

An abrupt manner in communicating to the patient the serious nature of his disease has, I am sure, a prejudicial effect upon most minds which, by inducing despondency, renders the sufferer indisposed to make the best use of means for his recovery. When the case is a grave one the patient should be urged to follow directions strictly, and warned that his recovery, so far as it is possible, depends upon his doing so. The relatives are the ones to whom the worst fears are to be confided. Here, as elsewhere, each case will have its particular bearings, and must be dealt with accordingly.

Prognosis in cardiac cases may appear simple enough, but it is not so in all. To alarm a patient on insufficient grounds is to make an impression on his mind that will not easily be removed—a condition inimical to mental repose. Patients who protest they would rather know the worst are, in my experience, those who bear bad news anything but well.

To sum up, it may be stated of rest that—(1) its place in the treatment of many forms and phases of heart affections is both invaluable and unique; (2) it is an indispensable preliminary to the adoption of anything of the character of "cardiac gymnastics;" (3) the duration of the rest is to be determined by the condition of the patient, especially by the extent of recession the dilatation has undergone, and the position and character of the apex-beat; (4) the effect of exercises upon the heart should be tested before deciding upon their extent or frequency; in all cases there must be sufficient intervals of rest between each exercise; (5) when the condition of a heart has improved under rest, but the strain of even moderate exercise is borne badly, massage of the limbs while the patient is lying down should be resorted to; in appropriate cases cycling on the level is one of the best forms of exercise in cardiac cases; (6) saline effervescing baths are useful in a limited class of cases, but they can never replace rest, though they may be beneficial as adjuncts to other treatment; (7) mental repose is invaluable, especially in patients of an emotional or neurotic temperament. An abruptly given bad prognosis may be most detrimental, and jeopardise any chance of improvement that may be present.

Mongolian Idiocy.—Muir ('Archives of Pediatrics,' New York, March) has investigated 26 cases, and discusses the condition generally. He found a history of drunkenness in 4 cases of his 26; 4 cases with either a family history of insanity or eccentricity; in 3 the father was violent in temper; in 1 the father was a suicide; in 4 the mother was neurotic and excitable; in 3 cases there were twins or triplets in the family; in 3 the intra-uterine movements were hardly felt; in 3 the mongol was premature; in 1 a previous child was anencephalous; in 8 the labour was prolonged or complicated; and in about one half the cases a statement of fright or worry was reported. Only 38 per cent. of the cases had tuberculosis in the parents, and only 3 had syphilis. In 15 cases the mother had bad health during pregnancy. Fifteen were males and 11 females. He describes the symptoms, appearances, and the data as to physical development of the patients. Fourteen of the cases were idiocy of the low grade, 4 medium, and 7 of a high grade. Speech came late. The best educated one knew his own name, could spell "Monday," and knew the year and month, knew all the figures and some of the letters. The prognosis seems bad in these cases. Treatment has been of no avail, and thyroid treatment was useless.—*Journ. A.M.A.*, April 4th, 1903.

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DEBATE

ON

DIABETIC AND NON-DIABETIC GLYCOSURIA.

Held under the auspices of the Chelsea Clinical Society at the Jenner Institute of Preventive Medicine, March 10th and 17th, 1903.

Dr. C. C. Gibbes, President, in the Chair.

(*Continued from page* 29.)

DR. ROBERT HUTCHISON: Mr. President and Gentlemen,—The subject of this debate is one of such wide interest that it must necessarily appeal to different people from very different points of view. It was dealt with at the last meeting from many different aspects; it was dwelt on from the chemical side, from the pathological side, and from the clinical side; and I have been asked by those who have organised the discussion to consider it to-night chiefly from the point of view of dietetics. Now from the dietetic point of view the distinction between diabetic and other forms of glycosuria, which I take it to be one of the objects of this debate to elucidate, is one of very great importance, because I think one may say that the non-diabetic forms of glycosuria are less likely to be influenced by the amount of carbohydrate in the diet than the true diabetic cases are. What, then, is the distinction between diabetic and non-diabetic glycosuria? Dr. Saundby, in his interesting paper, gave it as his opinion that the distinction was this: that if one could assign a cause for the glycosuria, then one was dealing with the non-diabetic type; and if one could assign no cause, then one was probably dealing with the truly diabetic type. I think one can hardly agree to that definition, because it must be in everyone's experience that one can often—or at least the patient thinks he can—assign a definite cause for the starting-point of the illness. Taking Dr. Saundby's definition, if a patient gets an injury to the nervous system, and this injury is followed by the appearance of sugar in the urine, one must say that he has a non-diabetic glycosuria. But I think it must be within everyone's experience that patients will often date the onset of what is undoubtedly a true diabetes from the occurrence of a nervous injury. I think the definition that Dr. Pavy laid down was one which is more in accord with clinical experience—namely, that those cases of glycosuria are to be regarded as diabetic in which the glycosuria is *persistent*. Dr. Pavy laid stress on the persistence of the appearance of sugar in the urine as the hall-mark, if one may say so, of the truly diabetic form of glycosuria; and to that I should like to add, that not only is the glycosuria in what I take to be the true form of diabetes persistent, but it is also *progressive*,—that is to say, the failure in assimilative power for sugar in true diabetes tends to become progressively greater, and the longer the disease lasts, assuming that the patient is not the subject of proper treatment, the less does his power of dealing with sugar and other forms of carbohydrate become. I think this point of progressiveness does distinguish to some extent the true diabetes of young subjects from the glycosuria met with in elderly people, especially those of gouty habit, for in the older subjects there is less of the progressive tendency. Such a patient passes a certain amount of sugar in the urine, and he continues for months, or possibly years, to pass about the same amount of sugar, even if he be not treated and there is no great tendency for his power of dealing with carbohydrates to go on getting progressively less than it was before. I am prepared to admit that that distinction is not absolute, because I think every one here must have seen cases of glycosuria in elderly persons which began in this way,—that is to say, began by being apparently not progressive, and then by-and-bye passed into the progressive form, and showed in various ways that they were now cases of true diabetes. So this distinction is one which is available only up to a certain point, and I do not wish to press it too far, or to say that progressiveness is always absent from a non-diabetic case of glycosuria. I question, indeed, whether one *can* lay down any hard and fast line between glycosuria and diabetes strictly so-called. I think from what I have seen that the distinction is necessarily rather an arbitrary one, and that one can never say definitely to which form a certain case is going to belong; it may appear at one time to belong to one type more than to another, and it is really only after the case has been in existence a long time that one can be quite sure in which category it ought to be placed.

There are, of course, many ways in which sugar may appear in the urine. It may appear, for instance, in consequence of failure on the part of

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the organism to compress the glycogen molecule into fat and store it up in that form. Again, sugar may appear in the urine in consequence of a deficiency of the glycogenic reservoirs; there may be insufficient storage-room in the liver and other situations where glycogen is usually accommodated. But I think insufficiency of storage-room is not likely to be a common cause of glycosuria, because everyone must have noticed how comparatively rare it is for patients with severe destructive disease of the liver to exhibit that symptom. I know Dr. Saundby has mentioned the occurrence of glycosuria in cirrhosis of the liver, but as a matter of fact one does not often see glycosuria even in patients who are the subjects of that disease in a marked and severe form; and as far as I know, in acute yellow atrophy of the liver, with rapid destruction of its cells, glycosuria does not occur at all. Nor am I satisfied that disease of the pancreas can often be a cause of diabetes in man. If one remembers that experimentally one has to remove the whole of the pancreas before sugar appears in the urine, one can hardly believe that disease of that organ is a common clinical cause of diabetes; because, were the whole of the pancreas so destroyed as to be deprived of its powers, one would inevitably find signs of failure to digest food on account of the absence of pancreatic secretion, and there would be the presence of fat in the stools. And though that sometimes does occur in diabetes, I think it will be admitted that it is extremely rare. If one assumes that it is only the islands of epithelioid cells in the pancreas which require to be destroyed in order to cause glycosuria, I think there is comparatively little true histological evidence for that view. It is difficult to see how one can get a disease of the pancreas which will destroy those scattered islands of cells and yet leave the rest of the pancreatic substance intact, or at all events capable of still performing the normal function of the organ. So, as far as I can see, we are still very much in the dark as to the true cause of diabetes. But what I wish to point out is that that does not necessarily mean that there need be any doubt as to how we should deal with cases of diabetes or of glycosuria from a dietetic point of view; for one of our chief objects in the treatment of any case where the glycosuria is persistent and progressive—that is to say, where it is of the truly diabetic type—must be to diminish the amount of sugar in the blood, or the *hyperglycæmia*, as it has been called. The dangers of hyperglycæmia were rather minimised by Dr. Hale White in his opening address at the last meeting. As far as I understood him, he seemed to imply that one tends to exaggerate the dangers and drawbacks of an excessive amount of sugar in the blood. Everyone knows the complications which are commonly said to be due to such hyperglycæmia. There are the suppurative complications, such as boils and carbuncles; there is gangrene; and there is a group of eye complications, such as diabetic cataract and diabetic retinitis. Then there are nerve complications, such as perforating ulcer, neuritis, neuralgias, and so on. And I gather—and I hope I am not misinterpreting Dr. Hale White—that he was inclined to think that these symptoms and complications are not so much due to the excess of sugar in the blood, but that they are part of a degenerative process of which the hyperglycæmia is merely another sign. But I think everyone must have seen cases in which some obstinate complication—let us say cellulitis, or gangrene, or possibly neuritis—has failed to clear up until one has removed the excess of sugar from the blood, after which quite a change has come over the picture of the local disease. I have seen cellulitis take on a healing action after the blood has been freed from sugar, and I have seen perforating ulcers in the same way start to heal up, although they had been in vain subjected to the ordinary treatment for a long time prior to the discovery of sugar in the urine. I think one must admit, in the face of observations such as these, that the occurrence of sugar in the blood is a bad thing, at all events in many cases, and that it does of itself tend to introduce complications into the course of the disease. Then I think the other great justification for the dietetic treatment, particularly of cases of persistent glycosuria, is, as I have said, its progressive character. It is just because they are progressive, because they tend to go on getting worse, because the tissues learn to assimilate less and less sugar,—it is on that account that the treatment of them is justifiable and necessary; for it has been shown pretty clearly in recent years that one can, so to speak, re-educate the tissues of such patients in dealing with sugar. I do not care what assumption one makes as to the pathology of diabetes; it does not matter whether you assume that it is an interference with the storing up of the

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glycogen, or the conversion of it into fat, or interference with the proper burning up of the sugar in the tissues; no matter which view is correct, or whether perhaps truth lies in all of them combined, —still I think the fact remains that to a large extent one can, by removing the excess of sugar in the blood, re-educate the organism in dealing with carbohydrates. The possibility of doing this was also emphasized by Dr. Pavy at the last meeting.

I think this loss of power in dealing with the sugar in the tissues can be to some extent hereditary; it does seem as if inability to cope easily with sugar were handed down in families; and I should like to know whether any one here has seen any good examples of that, and what their experience has been in taking precautionary measures against the onset of diabetes in the children of diabetic parents. I think that should be done more than it is. One should examine the urine of the children of diabetic parents from time to time as they grow up, and be on the look-out for the appearance of traces of sugar after the ingestion of a moderate excess of carbohydrates. One might then gradually educate their tissues to deal with carbohydrates better; or at all events one could see that the tissues were not flooded by excess of sugar, and that so their power of assimilation, which is hereditarily bad, was not made worse by over-strain.

Now the ease with which one can remove the sugar from the blood in a diabetic will be found to vary very considerably. There are some cases in which one has no trouble in removing all excess of sugar from the blood, and there are other cases where it is difficult or almost impossible to do so. The cases can be arranged, roughly speaking, in three groups. There is a group in which mere *limitation of the carbohydrates* is sufficient. That includes those which have been spoken of as the alimentary form of diabetes, in which it is assumed that there is some interference with the conversion of the sugar which is absorbed from the intestine into glycogen; and by merely limiting the amount of carbohydrate in the diet, in many such cases you can at once remove all sugar from the urine. Then there is an intermediate group of cases, where such mere limitation is insufficient; where you reduce the carbohydrates bit by bit, but you find that sugar still tends to appear in the urine, and it is only when you have *left carbohydrates entirely out of the diet* that the urine becomes sugar-free. That is a more severe type of case than the first I mentioned. Lastly, there is a third group of cases which are the worst of all, those where even *complete withdrawal of all carbohydrate from the food fails to remove sugar from the urine.* These are the cases referred to by Dr. Pavy as composite diabetes, and in them the patient is able to form sugar out of the proteid of the food. Now it must always be borne in mind that the amount of sugar which can be formed from proteid is very considerable, for 100 grammes of proteid yield 45 grammes of sugar. I think the practical lesson one has to learn from this is that proteids may be as dangerous to diabetics as carbohydrates are; and that is a point which it is worth while to lay some stress upon, because one does not read of it in text-books in the same way as one does about the dangers of carbohydrates. Yet there are some diabetics who seem to be able to form sugar even more easily out of proteids than out of carbohydrates. It is an interesting question whether a diabetic ever forms sugar out of the proteids of his own tissues. I do not think, in his review of the pathology of the subject, that Dr. Hale White dealt with this point. Can a patient break down his own muscle and form sugar from the proteid so obtained? If that does occur I think it must do so very rarely, because even in comparatively severe cases of the composite type, where a patient is forming a very considerable amount of sugar from proteid, one finds that if one withdraws from the diet not only all carbohydrates but all proteid also, and starves the patient for a day or two, the sugar will usually disappear from the urine entirely. I had a case recently of the composite type in the London Hospital where one found, even after the patient had been on strict diet for some weeks, that still a certain amount—about 20 grammes—of sugar was steadily excreted every day; but when the patient was put for twenty-four hours on a diet containing only meat extract, tea, and green vegetables and fat, a diet practically free from carbohydrates and proteid, the sugar disappeared immediately, although the patient during that time must have been breaking down some of his own body proteid. It would seem as if the sugar produced in the tissues were capable of more easy assimilation; and it is an interesting point whether such sugar may not be produced in the form of lævulose and

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not dextrose, lævulose being much more easily assimilated by many, if not all, diabetics than dextrose is. It follows from all this that the only nutritive constituent in the diet which is quite safe for diabetics is fat. One has to preach to these patients the importance of fat in their economy, and the necessity for their eating as much of it as possible; and I think there is no duty laid upon the doctor who manages a case of diabetes more important than to try to educate his patient in the consumption of fat. It is often a difficult matter, because I think your experience will agree with mine that unfortunately many diabetics are people who have difficulty in eating fat; they have never eaten much of it all their lives, but have consumed large quantities of carbohydrates, and have so overstrained their carbohydrate-assimilating function. And I think the great cheapness of carbohydrates in the present day may thus be to some extent accountable for the undoubted increase in diabetes. I notice constantly at the Children's Hospital how mothers tend to feed their children upon an excess of carbohydrate food; one sees dyspeptic troubles in these children which one recognises at once as due to the consumption of carbohydrates, their cheapness making them much more used than fats. I think one consequence of this may be that the carbohydrate-assimilating function tends to get over-strained, and so the patient becomes predisposed to diabetes. At all events, whether there be anything in that view or not, one has constantly to impress upon the patient, in all forms of glycosuria, the importance of fat in the diet, because that is the only thing which apparently cannot be converted into sugar.

If it be true that one has such different degrees of difficulty in ridding the blood of excess of sugar, I think one can deduce the third lesson, that there can be no uniform system of dieting suitable to all cases of glycosuria, but that each individual case must be taken on its own merits. Therefore one has, in any given case, to ascertain whether it is one in which there is no tolerance of carbohydrates at all; whether it is one of considerable tolerance; or whether, perhaps, it is one in which there is not only intolerance of carbohydrates, but in which the patient is breaking down proteid and converting it into sugar as well. In order to find out which group a patient belongs to, one must bring him gradually to a diet which contains no carbohydrates at all. I have said *gradually* because I think there is some risk in changing the diet of these patients suddenly to one in which there is no carbohydrate. I have myself seen several cases in which this sudden change in the diet seemed to precipitate coma. I remember not very long ago feeling rather unhappy about a man whom I put upon a strict diet rather suddenly because I was anxious to reduce the sugar in his blood rapidly, and who went straight into coma. It was difficult in that case to avoid the unpleasant conclusion that he would not have had coma if one had been more cautious in making the change of diet. I think, therefore, that it is important to proceed gradually. But there are many great authorities on the subject who are inclined to doubt whether the advent of coma is hastened by restricting carbohydrates in the food, and it would therefore be interesting to hear the opinions of members afterwards on the point, because it is a matter of great practical importance. My plan is always to proceed slowly in making a change, particularly if there is any evidence of excessive destruction of proteids,—that is to say, if there is a positive reaction with perchloride of iron in the urine. I was glad to hear Dr. Hale White at the last meeting emphasize the importance of the perchloride of iron test. It is a test which is easily carried out, and it is not carried out as often as it ought to be; I think it should be adopted as a routine measure in every case in which one finds sugar in the urine.

I know many people think it is impossible to keep a patient for any length of time on a strict diet, or at any rate that it is impossible to get a diet which is perfectly free from carbohydrate which patients will tolerate for any lengthened period. I think that is too pessimistic a view. I think everyone who can afford it—and every diabetic should be a millionaire, because it is an expensive disease—can get a diet which is tolerable for an almost indefinite length of time, because in recent years the construction of such a diet has been made very much easier than it was before by the introduction of casein preparations, which can be used in the making of breads and in many other forms. Casein is particularly suitable, because it does not so readily yield sugar as do other forms of proteid,—at all events, not by hydrolysis. Whether it does so in the human body I cannot say definitely. But there is good reason to suppose that it is less

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open to objection than other proteids, and it lends itself to administration in many shapes. Certainly the casein preparations have made strict diet for these patients very much easier. I have brought here for your inspection a bottle of sugar-free milk made from casein, which I always use; and, indeed, I do not know how one would get on without it in the strict dieting of these patients. It was remarked at the last meeting, I think, by Dr. Hale White, that milk can usually be allowed to diabetic patients in moderate amount. I cannot say that that has been altogether my experience. I have often found that even a pint of milk would increase the amount of sugar in a patient's urine, and that the other symptoms have at the same time increased quite markedly; I mean such symptoms as thirst. I am prepared to admit that lactose may be better borne than other forms of carbohydrate, but that it is a food which all diabetics will stand is not in harmony with my experience. Cases vary in that respect, and if you can eliminate lactose from their diet so much the better. I have found that sugar-free milk is a very great help; it can be cooked with eggs and so made into a custard, or it can be drunk plain, or with effervescent waters. It can also be taken in tea and coffee, and altogether it is a very great aid. Further, it has the advantage that one is able by its means to get down a considerable proportion of fat; it contains much butter-fat in a pleasant and digestible form. Then there is the question of bread. It was said, I think, by Dr. Saundby, that he always found starch in gluten breads, and with that statement I can entirely concur. I have examined many gluten breads, but I have never found one which did not contain starch. Moreover gluten bread is extremely nasty; it is a most unpalatable form of food, and I have entirely given up using it in favour of one of the breads containing casein. What I always use now is Callard's casoid meal bread, which is composed of casein and almond-flour and a little bran. It is practically carbohydrate-free—that is to say, it contains only about 1 per cent. of starch,—and it is one which patients can take almost indefinitely. I have had some patients on it quite recently, and I have never found a patient who has an objection to it. In connection with the bread, one would like to raise the question of toast. One constantly sees diabetic patients who say they never touch bread at all, they "only take toast;" and one finds that they have been recommended by a medical man to take toast, and they have been given to understand, or at all events they have understood, that toast is comparatively harmless for them. It would be interesting to know how that view arose, because it has always seemed to me that toast must be even worse than ordinary bread for diabetics; because all you do in converting bread into toast is to drive off a certain quantity of water, and I cannot see why the rupture of some starch granules and the conversion of a little of the outside layers of starch into dextrine can make any difference. I think this toast question is a pure fallacy, and that it is worse to allow patients to eat a given weight of toast than an equal weight of bread. At all events I should like to hear the opinions of members of the Society about this,—that is to say, whether they know of any reasons why toast should be considered better for these patients than bread. Much has been said by Dr. Saundby about the use of potatoes. Potatoes are, and must be, less harmful than bread for diabetics, simply because they contain considerably less carbohydrate; they contain, perhaps, only one third as much carbohydrate as a given weight of bread, and consequently one can recommend to patients a certain amount of potato as replacing a certain proportion of bread, and thus limit somewhat the carbohydrates taken. But that there is any peculiar merit in potatoes for these patients I cannot believe. Starch is starch, and sugar is sugar, no matter where it comes from, whether from the potato or from anything else; and all starch reaches the blood as sugar and must be assimilated and appropriated as such, and there can be no reason why the sugar which is derived indirectly from the potato should be more easily assimilable than that derived from other sources. The potash and other salts in the potato can have no influence whatever. One gets articles introduced every now and then like that which are said to be especially good for diabetics. Thus Dr. Donkin introduced the treatment of diabetes by means of skim milk. I should like to know what the experience of those present is of that plan. Personally I have never found it of use.

One must also mention alcohol. Dr. Saundby, in his remarks at the last meeting, rather gave us to understand that alcohol was a potent cause of diabetes, that it tended to produce glycosuria pretty commonly. I should be sorry if the impression

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got abroad that on this account alcohol is necessarily a bad thing for diabetics, because I think there can be no doubt that once a patient has got diabetes—that is to say, the genuine form,—alcohol is an extremely useful aid in feeding him. It is probably entirely burnt up by such subjects, and there is reason to suppose that, being burnt up, it helps to spare and economise fat. Alcohol has this further advantage, that it makes it easier for many people to digest a fairly large quantity of fat. One sees patients who find a difficulty in digesting fat, but who can manage to digest it if you allow them a certain quantity of alcohol. I am sure it is a useful aid in the feeding of these cases, and I should be sorry if it were supposed to be a thing to avoid merely because some people who get glycosuria have been indulging too freely in it.

Having got the patient on to a strict diet such as I have mentioned, one has to wait for a few days until one sees what happens. If one finds that the sugar entirely disappears, one must then add carbohydrate to the diet until one finds the point at which the limit of tolerance is reached. The best plan is to add bread in increasing quantities, one ounce at a time, spread over the various meals of the day, until one reaches a point beyond which the patient is not able to take an increased quantity without sugar reappearing in the urine. I had recently under treatment a patient æt. 28, who was suffering from diabetes of medium severity. He had a small tolerance for carbohydrates, about one ounce of bread. He was kept on strict diet for three weeks, and during that time his weight rose four and a half pounds, and he improved generally in every way. And then one found even after that short period of rest that the carbohydrate-assimilating functions were improved, and one was able to give him three ounces of bread without sugar reappearing in his urine. He left the hospital on a diet which contained less than three ounces of bread. One should not let a patient go on to the limit of his tolerance indefinitely; the patient should be given a permanent diet which is below the amount he can really utilise. Dr. Pavy made an interesting statement about the return of tolerance in those cases. He said that after one keeps a patient for a time on strict diet, such as I have mentioned, and has rested the carbohydrate metabolism, one finds the patient's power of dealing with carbohydrate returns; and he said you will know that by the patient losing weight and suffering from uncomfortable sinking sensations in the epigastrium. I should be sorry to contradict such an eminent authority on this matter, but I have difficulty in understanding how the symptoms which he described could arise. I have never observed them or heard of them from patients, and it is difficult to see how the mere increased tolerance could cause the patient to lose weight and have those uncomfortable sensations. I shall be glad to know if that has been the experience of other members of the Society, because if such symptoms occurred the fact would be of considerable use, as enabling us without making experiments to ascertain that the patient's power of dealing with carbohydrates was returning, and that so we might increase the amount of carbohydrate in his diet. Such, I think, is the way in which one should deal with permanent glycosuria, where one can remove all sugar from the urine by putting the patient on a diet which is free from carbohydrate. But one meets, especially in hospital practice, with that difficult group of cases which Dr. Pavy describes as composite, where even complete removal of all carbohydrate fails to rid the urine of sugar. What is to be done with those? I think that in these cases one should not be too strict in the matter of diet. There are two good reasons for allowing such patients a certain amount of carbohydrate. One is that many of them seem to form quite as much sugar from proteid, and possibly even more, as they do from carbohydrates, and therefore you do not gain anything by substituting proteid for carbohydrate. The other reason is that it is difficult to get patients to take enough fat if one has reduced the carbohydrate to a minimum; and in these cases the taking of fat is so important that one should allow them a certain amount of carbohydrate simply in order to act as a carrier of fat; one must make up one's mind to have a certain amount of sugar in the urine—say 60 grms. or so in the day, an amount which is not accompanied by any very great thirst or inconvenience, and which has only the disadvantage of being apt to give rise to some of those complications which I believe to be one of the results of continuous hyperglycæmia. These are the most difficult cases of all. Unfortunately they are almost the commonest also in hospital practice.

As regards diet in coma, one has to allow a

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certain amount of carbohydrate. The pathology of coma was discussed to some extent before, and I cannot go into it to-night, but there is no doubt it is due to excessive destruction of proteids, giving rise to acid substances, of which β-oxybutyric acid is the type; and by increasing the amount of proteid in the diet one runs the risk of increasing those acid materials, and so it is necessary to use a diet in which proteid is not too excessive. Also a certain amount of carbohydrate should be allowed; because even if you get sugar increased in the urine you do not necessarily do harm in such a case, for sugar is an admirable diuretic, and will tend to carry away a large quantity of the acid material which is circulating in the blood. So I think the best form of diet when the coma stage is reached is one of skim milk—and one says *skim* milk because one has a feeling that some of these cases may be due to fat in the blood; at least I have seen several cases in which the blood contained in large amount a substance akin to fat, which could be extracted by ether, and which made the serum almost milky. And hence it seems better to restrict the fat as well as the proteids on that ground. At the same time one can dissolve in the milk a large amount of bicarbonate of soda. My experience has been, however, that it is difficult, even by giving enormous doses of bicarbonate of soda, to neutralise to any degree the acid in the urine. I have in a recent case given three ounces of solid bicarbonate of soda in twelve hours by various methods—in milk, by the rectum, and injected under the skin,—and yet the patient at the end of that time passed urine which was decidedly acid. The amount of acid passed in the urine in a case of diabetic coma is very great, and it is very difficult, even by the largest doses of bicarbonate of soda, to neutralise it.

One would like to say something about drugs as an aid in removing sugar from the urine in these cases. I have dealt with diet, but as regards drugs I saw that Dr. Hale White asked in his address why opium was less used now. I have never been satisfied that opium does diminish the amount of sugar in the urine, and that is another point upon which it would be well to have an expression of opinion from different members. I have given opium fairly often in such cases, but I have never been satisfied that it made much difference. I am particularly doubtful about codeia being the best form of opium to use, and I shall be glad to hear about that from Dr. Hale White in his reply, because codeia has the great disadvantage in hospital work of being very expensive. I have often known an out-patient walk off with six shillings' worth of codeia pills, when it is practically certain that opium would have done as much good. I have seen cases where arsenic has done good, particularly, I think, in the glycosuria of elderly and gouty people. I saw, not long ago, two or three cases which occurred almost together in out-patient practice. They continued to pass a certain amount of sugar on restricted diet, and then one gave arsenic and had the satisfaction of seeing the sugar disappear from the urine. The arsenic was stopped, and the sugar came back. In all of them the sugar disappeared again when arsenic was given. I do not think it was merely a coincidence, but I have tried the same experiment repeatedly on other cases without any success. In the truly diabetic form of glycosuria I do not think that arsenic does any good at all, but it is a practical question which we might well discuss to-night.

Next I would like to ask, has any one present any experience in the use of thyroid feeding as an aid to removing sugar from the blood? Dr. Murrell published a paper some time ago, describing a case in which he had used thyroid feeding with marked success in removing sugar from the urine in diabetes. I have never felt myself justified in using it, because thyroid tends so much to increase the disintegration of the nitrogenous tissues in the body, a process which is already prone to go on too much in diabetes, and which one ought not to do anything to encourage. It is interesting from a theoretical point of view to discuss whether thyroid feeding makes any difference, because thyroid is the most powerful stimulant to metabolism which we possess; and, if the non-appropriation of sugar is in any way due to failure of metabolism, it is possible that the stimulating effect of thyroid might be beneficial.

Primary Tuberculosis of the Ovary.—J. E. Geurmell discusses a case of primary tuberculosis of the ovaries. Both ovaries were removed and found upon microscopical examination to be tubercular. There were no other signs of tuberculosis in any of the other organs; therefore the writer concluded that he had to deal with a primary lesion.—*American Journ. of Obstetrics*, April, 1903.

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A CLINICAL LECTURE

ON

EFFUSIONS INTO THE KNEE-JOINT.

Delivered at Westminster Hospital.

By E. PERCY PATON, M.D., M.S., F.R.C.S.,

Assistant Surgeon to the Hospital.

GENTLEMEN,—I have chosen as my subject this afternoon effusions into the knee-joint as being a common affection, and one for which many different plans of treatment may be necessary. As, however, it would be impossible to deal completely with the subject in the time at my disposal, I shall restrict my remarks to *chronic* effusions into the joint.

In the first place a few words may be said on the detection of fluid in the knee-joint. As a rule this is a very simple matter, but, although this is so in most cases, one finds a good many mistakes are made. One notices that many men are content to examine a knee-joint in a somewhat perfunctory manner in relation to fluid; the examiner is not careful enough to accumulate the fluid which is in the joint underneath the patella, so as to produce the riding of that bone which is the most characteristic sign. Unless this is done by placing the hands one above and one below the patella, and so accumulating the fluid from the superior and lateral pouches beneath it, fluid may not be detected even when present in fair quantity, and particularly so in a joint which has had fluid in it for a considerable time. The capsule is then relaxed, and the patella may be in contact with the femur, when there is a great deal of fluid in the lateral pouches. On the other hand, you frequently hear people tell you that there is fluid in the joint because they have violently attempted to elicit signs of its presence; they have lifted the patella up and brought it rather roughly down against the femur; thus they get a tap, but it is not the tap which tells of the presence of fluid. Another mistake is

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sometimes made when the joint is full of soft granulation tissue. This is an error of considerable importance. It is not very frequent, because the conditions under which you find a joint distended with soft granulation tissue are usually such that you can easily detect it for other reasons. But it is important to remember that if you have *fluid* in the knee-joint, you ought to be able to get a distinct *tap* of the patella down upon the femur. Otherwise, where there is a good deal of granulation tissue you can feel the patella going down, but it does not strike the bone below. Again, there is one other condition in which you do not get the patella striking the condyles; and that is when there is great tension. This is much more common in acute than chronic effusions, and in this condition you cannot get a patellar tap: but you can determine the presence of fluid in a case of this sort by getting a thrill through the joint, and from the free fluctuation in all directions.

We next come to the classification of *causes* of chronic fluid in the knee-joint. The most important division to make is between those which are *traumatic* in origin and those which are dependent upon some *chronic disease* in the joint. Those cases which are traumatic are in their early stages usually of the acute kind, but after a short time has elapsed the acute signs disappear, and you are very often left with a chronic effusion into the joint as a result of the injury. Such chronic effusion may be dependent upon fracture; it may be dependent upon simple wrenches of the joint, various twists and strains, such as occur playing tennis, or by sudden recovery of one's balance under difficult circumstances. A loose body or a loose cartilage may also be responsible. In the cases in which fracture is responsible for the effusion, and in the cases in which the joint is filled with fluid as a result of a wrench, there may be, in addition to the ordinary inflammatory fluid, a large quantity of blood. This blood sometimes takes a long time to disappear, the blood clots, and it is then not readily absorbed. In many cases where there has been a simple wrench or twist of the joint the fluid is simply serous, with a small amount of blood mixed with it. But in some even of these cases there may be a large quantity of blood, and that blood in itself is sometimes responsible for a considerable prolongation of the case, so that a previously acute condition becomes a chronic one. The cases in which there is a loose body or cartilage in the joint are frequently those in which the primary trouble has been a wrench. But sometimes a patient complains of effusion into a joint with no clear traumatic history; the effusion does not clear up, and a loose body or cartilage is really at the bottom of the trouble. In any case where there is reason to suspect such to be the condition one must inquire very particularly into the history, in addition to very carefully examining the joint. There are certain causes for the conditions I have referred to passing into the chronic stage which are within our power to prevent. Probably the most common cause of this chronicity is want of rest after the injury. Often the patient does not think very much of it at the time; the pain is not severe enough to compel him to lay up, and the consequence is that he still gets about on it; so that a joint which, if treated by rest for a few days, would have rapidly become cured, goes on to chronicity, and further fluid accumulates in it, and a very considerable amount of treatment is necessary before the trouble subsides. Another group of cases allied to these is where some rest has been given, but it has not been long enough. A class of case which is also very important, is where there has been too much rest given to the joint. In many traumatic cases, after the lapse of a certain time, it is not wise to keep the joint fixed in a definite position. The cases here referred to are those of injury which have been kept for some time at rest, in which the synovial membrane of the knee-joint is affected with simple inflammation; there are many folds in this membrane, and if these folds lie in apposition for a long time adhesions may form, which, however, are not very firm unless suppuration has taken place. In these cases, when the patient begins to get about he experiences pain, and so to avoid the pain he does not move the joint, but keeps it stiff; and as, if he walks more than a very short distance, the result is a return of the effusion, the leg is again put on a splint and kept longer at rest, and so the case, instead of improving, remains *in statu quo*. This is the class of case which sometimes gets into the hands of irregular practitioners, who violently move the joint and cure the condition at once by breaking down the adhesions. In another group of cases the patient is kept at rest, because immediately on moving about he gets a slight

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return of the effusion. This, however, usually subsides again shortly, and it is not a circumstance to be taken too much notice of at first. In traumatic cases which remain chronic it must be always borne in mind that there may be some local specific cause at the bottom of the trouble, some cause which is really responsible for keeping up the collection of fluid. For example, there are a certain number of cases which begin as an ordinary traumatic synovitis; they subside almost at once as far as the acute symptoms are concerned, and then the fluid remains chronically for weeks, and various remedies are tried without any success. At the root of these cases there is sometimes tuberculous trouble. It is a curiously interesting fact that tubercle very rarely commences in a joint which has been the subject of a *severe* injury. It is very rare indeed for tubercle to commence in a joint which has been dislocated and reduced. On the other hand, tubercle not infrequently begins in a joint which has been slightly injured or sprained. This fact is borne out by experiments upon animals, in which it has been shown that if tuberculous material is injected into an animal, and then a violent injury is done to a joint, it is very unusual for that joint to become tuberculous. But if you wrench a joint and produce an injury similar to a strain in the knee, not infrequently, and in fact almost always, it becomes infected by tubercle. The explanation of this is not very clear, but it is a fact which is borne out by experiment in a large number of cases. So that tubercle may be at the bottom of the long continuance of chronic effusion into a joint in some instances.

But one must not only look to local causes for prolongation of effusion, but also to the general condition of the patient. If the patient is rheumatic, or in a general condition of ill-health, or has recently suffered from syphilis, any of those conditions may be responsible for keeping up the local effusion.

We now come to that large group of cases which are not traumatic, but in which the effusion is due to disease. Of course, in a very large number of them the effusion commences acutely, as in cases of septic infection of a joint; but in quite a considerable proportion of cases you see a chronic effusion commencing practically without any pain, or with very little pain, and practically the only evidence you have of the disease at first is the presence of fluid. Of such cases one of the most important causes is *osteoarthritis*. I am bound to confess that it is very difficult to say exactly what is meant by osteoarthritis. Almost certainly under the term osteoarthritis a large number of different kinds of disease are at present grouped, which ultimately may be isolated from one another. At any rate osteoarthritis includes certain changes in joints which are well known, and it is a convenient term to use. Very often in chronic effusion into the knee-joint you find that osteoarthritis is responsible. In the out-patient room one sees probably two cases of effusion into the knee-joint for which osteoarthritis is responsible to one of any other variety. So that it is a very important class. Another cause is *gonorrhœa*, although the effusion here is not of such a marked character as in some other diseases.

Then *syphilis* is responsible for a few of the cases, and the syphilitic cases are very often overlooked because they are not expected—partly, I think, because there is not very much written in the text-books upon the occurrence of effusion into the joints, particularly the knee-joint, in connection with syphilis. I do not mean to say it is very common, but as a fair number of cases occur, and as they are obstinate to treatment unless the true cause is recognised, their recognition is important.

Tuberculous disease sometimes commences with chronic effusion into the joint.

Then there is the series of cases which are due to some sort of *infection*, such as the cases which have only recently been brought to light by the researches of Poynton and Paine and others, in which there is a diplococcic infection of the joint. They are not clearly understood at present, and perhaps it is not well to lay too much stress upon the pathological aspect of them. But there is effusion into the joint, often without much constitutional disturbance, lasting for some length of time, for which some sort of infection is responsible. They appear allied on one hand to osteoarthritic cases, and on the other hand to the more severe cases of infection. Some chronic effusions also occur in which *anæmia* apparently is the main cause. It has been pointed out by Bennett that in a certain number of cases in girls of sixteen or seventeen years of age you find passive effusion into the knee-joint, associated with very little pain,

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and the only other things present in these patients were marked anæmia and perhaps menstrual irregularity. What association there is between the conditions it is impossible at present to say.

The next point which is of interest is, how to distinguish these different causes from one another. The history may be of great importance, as, for example, sometimes the chronic effusion follows an acute rheumatic inflammation. Of course there is no difficulty in determining the cause in a case of that sort, although why the effusion should remain it is very difficult to say. Again, in gonorrhœal cases the history may be of assistance, although the history one gets cannot be relied upon. The same applies to syphilitic cases to some extent, although not to a great extent. In many instances the history is quite useless, and does not throw any light upon the condition at all; and then one must rely in the first instance upon careful examination of the joint to determine any other features which may be present in addition to fluid. And then a careful investigation must be made as to the general condition of the patient, for in this something may be found to throw light upon the effusion. In examining the knee-joint in detail it is always important to bear in mind the different parts which enter into the formation of the joint. One so frequently sees, especially amongst students, that important points are overlooked because there is no method about their procedure. But if one bears in mind the different parts of which the joint is composed, and if each of one's manipulations is directed towards eliciting a certain sign or fact, one will be much nearer the diagnosis. In this way you are more likely to get at all the information about a joint than in any other way. Your examination should be first directed to ascertain whether the synovial membrane is diseased, then as to whether the bones are diseased, and then one should examine the cartilages and ligaments and surrounding tissues. The most distinct evidence of synovial disease is the existence of effusion, and in that case one has no difficulty in determining that there is some disease of the synovial membrane. As to the sort of synovial disease which is present, you may get some assistance if you can find that there is any thickening of the membrane, and especially if this thickening is located in any one particular spot. A mass in one particular place associated with effusion would be exceedingly suggestive of syphilis on the one hand, or of tubercle on the other,—that is to say, tubercle commencing in the synovial membrane. The movements of the joint should be carefully tried with a view to determining the condition of the cartilages and bones. It also sometimes gives you a good deal of light as to the condition of the synovial membrane; for instance, when you get a considerable amount of soft grating and crepitus, this is a feeling in the joint which is indicative of considerable hypertrophy of the fringes of the synovial membrane. This may be due to osteoarthritis, or it may be due to a considerable amount of deposit of fat in the synovial membrane; in most cases it is dependent upon the fringes of the synovial membrane becoming thickened, and having small cartilaginous bodies in them, giving rise to that creaking which you obtain under the hand in a joint when it is moved. In the knee-joint a careful examination of the patella will sometimes throw light on the question as to whether there is any increase in the size of the cartilage, or any spreading of cartilage over the edge of the bone in cases of osteoarthritis. The patella in those cases is usually broadened, and sometimes exceedingly markedly so. Lipping at the edges of the femur is much more easy to determine than it is over the tibia, and it is better to examine the joint in the flexed than extended position if possible. In tuberculous cases, when the trouble begins with an effusion, beyond some thickening of the synovial membrane it is sometimes very difficult to make out anything else; very likely in such a case you will find nothing further by simple examination of the joint. In gonorrhœal cases the amount of effusion is generally small; effusion is not at all a marked feature in gonorrhœa. In syphilitic subjects syphilitic joint disease may occur either during the secondary or during the tertiary period. But the chronic effusions are most usual during the tertiary period, and in them you sometimes find gummatous deposits, but very often you find nothing beyond fluid. One of the most characteristic features of the syphilitic cases is the amount of pain they sometimes get with very little to show for it. There is no redness, and practically no increased heat; there is very little pain on movement generally, and yet at night they get a good deal of pain in the part. Another rather characteristic feature, which is more common in

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hereditary than in acquired cases, is the bilateral symmetry of the disease. You very often find that in cases of syphilitic effusion into the joint both knees are affected, and in the hereditary cases you find they are exceptionally painless, and yet the amount of fluid may be very considerable. Many observations were made a year or two ago on the occurrence of knee-joint disease occurring in patients who are the subjects of interstitial keratitis. In all cases seen with interstitial keratitis the knee-joints were examined, and it was found that in a large number of them there was a considerable amount of fluid present in both knee-joints, although the child did not complain of them. The effusion cleared up very rapidly under the treatment which was being given for the interstitial keratitis.

With regard to the general examination of the patient, of course a routine investigation should be made of the organs in the chest, particularly of the heart, with the view of determining the possibility of the case being rheumatic at the bottom. The other joints should also be carefully examined. In a large number of cases this will throw considerable light on the nature of the trouble by showing that some of the other joints are markedly affected with osteoarthritis.

The question of any discharge from the urethra or vagina should be carefully gone into. Many cases are overlooked which are really gonorrhœal because no such inquiry is made. Any signs of past syphilitic lesion must also be looked for. The general condition of the patient must not be forgotten, because sometimes the presence of anæmia or some other poorness of general health may be responsible for the onset of the effusion, or for its continuance.

In examining some of these joints full of fluid, particularly the knee-joint, one sometimes finds a complication which is interesting—namely, the development of *cysts* in connection with the synovial membrane. This is perhaps more commonly seen in association with chronic senile osteoarthritis than in any other variety of disease, although it occasionally occurs in tuberculous affections, and also sometimes in other diseases. For example, I saw a patient within the last few weeks with an enormous cyst extending down the back of the calf, and reaching so near the surface that it was easily translucent, and I was afraid of it breaking through the skin. In this case, on careful examination, one found the cyst to have a distinct connection with the joint, which showed some signs of early osteoarthritis. One of the most curious features associated with many of these cysts is the small amount of fluid that is present in the joint, and the large amount of fluid there is in the cyst. It seems perfectly clear that in such a case the fluid is formed partly in the cyst, although this has commenced in connection with the joint. Sometimes these cysts are of enormous size. Four or five years ago I saw one which was as large as a cocoanut. It was supposed to have no connection with the knee-joint. It was operated upon and was ultimately removed. At the first operation no opening was found communicating with the knee-joint; but the fluid re-accumulated, and then a connection with the joint was found. Of similar nature are those cases in which a cyst is first noticed at a long distance from the joint; they have been described fully by Morrant Baker some years ago. They present the following features:—A fluctuating swelling is found at some distance from the joint, for example down the calf of the leg. It is thought to be a chronic abscess, and unless one is very wary it may be opened without proper precautions, and a blow up follows in the joint. Nowadays, of course, more care is taken, and a chronic abscess would not be cut into without first preparing the skin and the instruments and the hands, so that the amount of damage done if a mistake in diagnosis is made is not likely to be very great. But in the old days, when an opening was made without any precautions, the result was most likely to be severe trouble in the knee-joint from the casual opening of one of these cysts. The mistake is recognised by the escape of oily synovia instead of pus.

We will now turn to the question of *treatment*. In chronic effusion into the joints, where the effusion is excessive, undoubtedly the best thing to do, after it has been present for some time, is to draw off the fluid. Of course the knee-joint must not be tapped unless every precaution is taken to prevent infection; but there is no difficulty in doing this, for you can take time to prepare the joint thoroughly, and then there is no danger attached to drawing off the fluid. Most usually the needle is inserted at the lateral aspect of the patella, but for many reasons it does not seem to be the best

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place to tap the joint. If one examines an ordinary section of the joint one finds there are folds of synovial membrane which pass up into the notch between the condyles; and one has seen cases in which there has been an aspirating needle passed in this direction and yet no fluid has been drawn off, because the point has become engaged in one of the folds. If, however, you pass in the needle at a point on a level with the upper border of the patella, downwards and inwards or downwards and outwards, it is not likely to engage any fold of synovial membrane, and the needle is not likely to be blocked, as its point will lie just behind the patella. One has so often seen an attempt made to aspirate a knee-joint and it has been unsuccessful that I think it is rather important to see that it is done in the right situation. When the fluid has been drawn off, very often, by means of a simple firm bandage, or a Martin's rubber bandage, the fluid may be prevented from re-collecting. This is particularly so in traumatic cases which have hung fire. These cases may have been treated for the ordinary acute trouble, and the fluid remains. If there is still a large quantity of fluid in the joint after ten days or a fortnight, you can tap it, put on pressure, and keep it at rest for about a week, often with the best results; then gradual movement and massage may be begun. In those cases where the fluid is not excessive in quantity it is not worth while tapping. The result of counter-irritation is quite as satisfactory, if not more so. Many people decry the use of iodine for this purpose. Of course I do not think that once painting with iodine is any good, but in the out-patient department one finds iodine very useful. If you instruct the patient to get some Liquor Iodi and paint it on night and morning for several days until it is sore, you produce the same effect as with a blister, and without its inconvenience. I have found this a very useful plan of treatment. Painting it once and letting the patient go away does not appear to have any effect, but used in the way I have mentioned it produces a powerful counter-irritating effect. The blister is perhaps the best counter-irritant if the patient is laid up. If you can put a blister on each side of the joint, and in a couple of days one above and below, you will generally by that means get rid of the fluid in about a week. Of course there is the more active plan of counter-irritation by using the actual cautery, but unless the case has hung fire for a long time that measure is not necessary. In obstinate cases it may be well to use Paquelin's cautery at a dull red heat, especially where there has been a good deal of pain. But in most of these chronic cases there is practically no pain, and it is therefore unnecessary to treat them in this way. But putting aside these methods of counter-irritation, undoubtedly one of the best plans of treatment of cases which fail to settle down after a rest of ten days or a fortnight is treatment by massage. Suppose you have an acute case, which has quieted down as far as the acute symptoms are concerned, and now there is a little fluid left which will not clear up. If you begin massage you will very often find that the first effect of it is to increase the quantity of fluid—say for twenty-four to forty-eight hours. I always tell patients to whom I recommend this treatment that they will find the fluid larger in quantity at first, but that that is not an indication that the massage is doing harm. Until it has been used for three or four days you cannot tell what the effect of it will be in any given patient. In many cases it has a most marked effect in getting rid of the fluid and relieving that patient from the weak condition of knee. These patients often cannot walk well, not because there is anything wrong with the joint, but the muscles are flabby and have not been used. Massage gets them into condition, and thus materially assists to prevent them getting wrenches and twists, which would cause a return of the fluid directly they begin to get about again.

There are other methods of treatment which have come into vogue very much lately—namely, by hot air baths and electric light baths. There can be no doubt that, under proper supervision, the hot air treatment is in many cases exceedingly good; the fluid will disappear rapidly, and pain will be relieved; the suppleness of the joint will return, and the patient will quickly get better. Sometimes one can utilise as a makeshift in the treatment by hot air an apparatus intended for giving hot air baths in cases of Bright's disease. This is a pure makeshift, and is not the same in effect as the proper apparatus. You cannot get so high a temperature, and the result is not nearly so good. But in some cases where you have not any other apparatus at hand some benefit may be got by its use. When you have tried these and other plans of counter-irritation to which I might refer, such

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as the traditional Scott's dressing, one is left with a residuum of cases in which there is still fluid in the joint. You have drawn off the fluid, and treated the case in various ways, but the case does not mend. The time has now come for an exploratory operation, and this offers several advantages under these conditions. In the first place it enables you to make a definite diagnosis. The fluid if preserved may enable you to determine the presence of micro-organisms of some sort in it. Tubercle bacilli are very rarely found in these cases, but inoculation experiments with the fluid may give positive results. Very often you can, by operation, find thickened fringes of synovial membrane which are clearly responsible for the trouble. These should be removed. You can sometimes determine that the case is osteoarthritic, and I have seen great relief follow in cases of this sort in which there has been long-continued effusion, upon merely opening the joint, putting in a drainage-tube for a week or ten days, and sewing up the wound, and then allowing the patient to get about and have massage. This has enabled patients to be freed from their trouble for long periods. In one case I can remember the trouble was not thought to be osteoarthritic at all, and there was no reason for thinking it to be such. It was one of those indeterminate cases in which prolonged treatment had failed to give relief, and the plan I have just related was successful in relieving the patient, though the case was really, as the operation showed, osteoarthritic. Any loose bodies which are found can be removed; and if a loose cartilage be found it can be dealt with in the ordinary way—namely, by stitching it or by its removal.

But even then there are a few cases which are left at the end of all this investigation and treatment, cases in which you cannot determine that the disease is tubercle; there seems to be no osteoarthritis, there is apparently no rheumatism, and evidently the general condition otherwise of the patient is good. Very rarely it may possibly be advisable to suggest converting the joint into a stiff one, but this is very exceptional, and it should be recommended only in those cases in which the effusion has gone on for a very long time, and the joint is so loose that it is practically of no use.

The plans of treatment which we have been speaking of so far have all been local ones. There now remains to be mentioned the general treatment. If there is a constitutional defect in the patient you must treat this; but general treatment should be carried out even if no constitutional taint exists, such a trouble as anæmia or other obvious malady being specially dealt with. Sending the patient away for a time to the seaside may have a markedly beneficial effect in an ordinary case which has gone on for weeks,—that is to say, such a case as a traumatic synovitis which has refused to respond to ordinary measures. Syphilitic cases naturally remain obstinate until, under the suspicion of their nature, you give iodide of potassium and mercury; and for osteoarthritic cases the drugs given are legion, in addition to various baths, waters, etc.; but the discussion of these forms of treatment would carry us too far.

A CASE
OF
TUBERCULOSIS OF THE BREAST IN A PATIENT ÆT. 73.*

(Under the care of Mr. MARMADUKE SHEILD.)

THE following case is worthy of record, owing to the extreme rareness of mammary tuberculosis in elderly women. In Delbet's collection of thirty-seven cases, the oldest patient was fifty-two years of age, and only six of the patients were over forty.

A woman æt. 73 was sent up to St. George's Hospital with a tumour of the breast, and was admitted under the care of Mr. Marmaduke Sheild on January 22nd. She was a cook by occupation, and had never borne children. Her attention had been drawn to the tumour in the right breast two months previously by neuralgic pain, and she then discovered a small nodule in the extreme outer portion of the breast. This nodule had steadily increased in size, causing a moderate amount of pain. There was no history of injury; the patient

* For the notes of this case we are indebted to Mr. C. English, F.R.C.S., Surgical Registrar to St. George's Hospital.

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had always been a healthy, active woman, and there was no family history of tubercle.

On admission she was a fairly vigorous subject, with some nasal catarrh and bronchitis. In the upper and outer quadrant of the right breast was a rather tender rounded tumour about 1¼ inches in diameter; quite hard and resistant at the centre, and more indefinite at the periphery; not adherent to the overlying skin, and movable on the deeper parts. The nipple was slightly retracted, and there was no discharge. In the axilla two or three enlarged, hard, but movable glands were felt. The lungs were carefully examined by Dr. S. Vere Pearson, medical registrar, who reported that there was no evidence of pulmonary tuberculosis.

On February 2nd Mr. Sheild made an incision into the swelling and found that it was a chronic abscess containing about ʒiss of curdy, yellowish pus. The wall of abscess cavity was about ⅓ inch thick, and was surrounded by an area of induration. A piece was excised for microscopical examination, and a drainage-tube inserted into the cavity, the wound being partially closed with stitches.

The excised portion was examined by Dr. Rolleston, and was found to contain typical tuberculous granulation tissue. There was no breast tissue present, but, on the outer part of the section, portions of the surrounding fat and of the pectoral muscle were seen, the latter being in a condition of chronic interstitial myositis. Internal to this was granulation tissue containing well-developed giant-cells; the inflammation was of various stages, in parts being obviously of some standing, and small areas of caseation were present.

The drainage-tube was removed on February 6th, and the wound had healed on the 10th. A further collection of pus, however, formed, and on the 18th a second incision was made; the abscess cavity was thoroughly scraped out and swabbed with pure carbolic; gauze plugging was inserted, and the wound slowly granulated up.

The patient was seen on March 30th; the sinus had then healed, and the condition of the axillary glands was unaltered.

DEBATE

ON

DIABETIC AND NON-DIABETIC GLYCOSURIA.

Held under the auspices of the Chelsea Clinical Society at the Jenner Institute of Preventive Medicine, March 10th and 17th, 1903.

Dr. C. C. Gibbes, President, in the Chair.

(*Concluded from p.* 48.)

Dr. Hector Mackenzie: Dr. Hale White in his opening address put forward many most interesting and difficult problems, some of which have been considered by previous speakers. The subject is a very extensive one, and I propose to confine my remarks to the consideration of some of the non-diabetic forms of glycosuria. These are of much clinical interest, and the problem as to whether a case with glycosuria is one of diabetes or not is constantly coming before us for solution. In the first place there are the cases of what one may call pseudo-glycosuria, which depend upon some error or fallacy in the test. Dr. Hale White in his opening remarks drew attention to possible sources of fallacy in the test for sugar commonly employed. A little sugar is a dangerous thing to diagnose with certainty, and yet one finds not infrequently medical examiners in their reports for life insurance, on the strength of a slight change in colour of Fehling's solution on boiling, assert the presence of a trace of sugar. Sometimes the Fehling's solution is at fault; sometimes the reduction is due to glycuronic acid, creatinin, uric acid, or some such substance. To avoid errors we must first test the Fehling's solution by boiling with an equal volume of water for two minutes. If the solution is in good condition no change will take place in it. Next we must see that not more than half a volume of urine is added to the Fehling's solution, and that the mixture is only brought to the boil. Almost any concentrated urine, if boiled with Fehling's solution for some time, will produce a change in colour. If the test has been properly carried out and we only get a greenish yellow colouring, we cannot say whether sugar is present or not without further tests. I have notes of four cases which have

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occurred during the last few years where medical examiners have reported slight traces of sugar. In three of these everything else was favourable, but the cases were not proceeded with. The other eventually went to another office and was accepted at ordinary rates. The rule I would advise is to get the applicant to supply one or two further specimens, and if these are normal the so-called trace may, I think, with safety be ignored.

I should like to impress on all medical examiners for life assurance that it is never safe to assert the presence of a small quantity of sugar in the urine on the strength of a single test.

Dr. Pavy, with his usual lucidity, has given us a definition of diabetes which, if we keep it before our minds, will materially help us to differentiate glycosuria due to diabetes from that which is not. Diabetes is a more or less persistent impairment of the assimilative action for carbohydrate food. It is this greater or less persistence of glycosuria which, according to Dr. Pavy, constitutes diabetes. As cases come before us in practice they may be divided into three classes. A person comes to us because he is ailing. He presents certain features and complains of certain symptoms which lead us to expect to find sugar in the urine, and we do find it. He is wasting, he is losing strength although he eats well, and he is passing a large quantity of urine. We can positively assert that he has diabetes, and we know that we shall find that sugar is more or less persistently present in the urine. We know from the history of the illness combined with the results of our examination what is the true nature of the case. Another person comes because he is out of health in some way, but with no symptoms of diabetes, and we discover in our routine examination that there is sugar in the urine. In this case the glycosuria may be temporary or persistent. It may be a symptom of another malady, or it may be the first sign of impending diabetes. We must wait and watch before we can express an opinion. Another person comes not because he is ailing, but because he wishes to insure his life. He says he is in perfect health, but we examine the urine and find that it contains sugar in greater or smaller amount. Here we can but note the fact that sugar is present; we must make further investigations to enable us to say what it means. It is only by means of repeated examination that we can determine whether there is any more or less persistent impairment of the assimilating power for carbohydrate food.

The assimilating power of all of us, even in health, is not unlimited. We know in all of us even in health sugar will appear in the urine if undue amounts of various kinds of sugars are administered. Six to eight ounces of glucose, four ounces of lactose, and intermediate amounts of cane or fruit sugar will cause the corresponding form of sugar to appear in the urine. It is impossible, however, in the normal subject to produce glycosuria by the administration of starchy food.

Interesting as they are, I must pass over those various forms of glycosuria which are non-diabetic because non-persistent, being produced by some temporary cause. The glycosuria due to asphyxia, as in whooping-cough, coma, convulsions, due to lactation, due to certain poisons, as carbon monoxide, phloridzin, preparations of thyroid or suprarenal gland, I shall not consider. Neither shall I dwell on glycosuria or diabetes as connected with acromegaly, exophthalmic goitre, or gross cerebral lesions, such as tumours of the medulla or in the floor of the fourth ventricle.

I wish more particularly to consider those cases of glycosuria which are of a temporary and transitory character, which approach the borders of true diabetes, and always require special thought and consideration.

Accidental glycosuria.—In insurance work, and more rarely in medical practice, cases of glycosuria are met with which appear to be of an accidental character. I remember a patient, a middle-aged man, who was sent to me for an opinion a few years ago by his own doctor. The man, who was a little over weight, felt out of condition and was easily tired. His pulse was slightly intermittent, and his doctor suspected a deposit of fat on the walls of the heart. On examination I found that the urine contained abundant sugar, and I thought this might explain matters. I wrote to the doctor giving him my views and drawing his attention to the urine, but I understand he never found any sugar, so I presume the presence of sugar when the patient visited me was accidental.

The following case is a good illustration of accidental glycosuria as met with in insurance practice.

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An old patient of mine, a man of forty, formerly resident in London, but now settled in a large provincial town, where he holds an important responsible post, wrote to me one day, about two years ago, as follows:—"I should very much like to have your advice. On Monday I was examined by a doctor to an insurance company. He tells me privately I have sugar in my water. I am sending you a small bottle. I should be much obliged if you would report on it for that or other things. If you confirm the doctor's statement, will you be kind enough to tell me exactly what it means in the present and in the future, and also what diet is best? I have not been taking much exercise lately, and I have been rather costive. I am enclosing a list of the things I eat, so you can judge. If you think I had better do without any particular articles, could you delete them and return this list altered? I shall be most anxious to hear what you think."

I knew my old patient to be of good constitution and to be a most careful liver, and I had always found him organically sound. I was sorry to hear that sugar had been found in his urine, but I was relieved, on examining the specimen he sent me, to find it to be quite normal. I accordingly wrote and told him not to be alarmed, as the specimen he sent was healthy, and the presence of sugar in the urine might have been quite accidental. I said I did not wish him to alter his diet in any way, but to send me a number of samples, mixtures of urine passed at different times of the day. He did so, and on examination I never found any trace of sugar. In a subsequent letter he wrote: "It may interest you to know that last Monday morning I called on the doctor who refused to pass me, and requested him to make a further test then and there, and he was unable to find any sugar. He refuses, however, to alter his opinion about the specimen he first of all examined." My patient sent me other specimens, which also proved to be normal. He then wrote: "I told the insurance company I was willing to be re-examined by any doctor they chose, but they think that one examination would not be sufficient; in fact, they talked of several tests at lengthy intervals." I advised him in these circumstances to apply to another office, and to mention exactly what had happened. This he did; he was examined by another doctor, and the examination proved satisfactory, and the life was taken at ordinary rates.

The second point, then, I should like to impress on the medical examiner is that it is possible that a person may have sugar in his urine on one or more occasions without having diabetes.

The following were probable instances of accidental glycosuria:

I was asked to examine on behalf of my insurance office a gentleman sixty-five years old, of no occupation, who had only a few days before been examined by Dr. X—, of the A. and B. Office, and accepted as a first-class life. The family and personal history were good. Height, 5 feet 9¼ inches; weight, 13⅛ stone. As he had been so recently examined, the proponent objected to a second complete examination. I made my examination after lunch and found that the urine contained a good deal of sugar. I reported that without further examination I was unable to estimate the risk, and nothing further was done in the matter.

The converse to this case was one which was examined by my friend Dr. George Johnston, and sugar being found, the proponent was asked to attend again. Meanwhile he went to another office, and the urine presumably being found to be normal, he was accepted at ordinary rates. The man had had port at lunch before the first examination, which was very likely the cause of the presence of sugar in the urine, just as the lager beer and the liqueur at lunch were the cause of it in the case Dr. Pavy mentioned.

If sugar be present more or less persistently, but only in small quantity, and there be no symptoms of diabetes, I think from a clinical point of view it is preferable to call the case one of glycosuria. Dr. Pavy mentioned a very interesting case of that kind where slight glycosuria, after persisting for some time, disappeared and remained absent for many years, to return again in a slight form. Loeb has recorded a similar case where a woman for four and a half years excreted sugar in her urine in quantities varying from 0·22 to 0·4 per cent. At the end of this time the urine was absolutely free from sugar, and remained so even on the ingestion of large quantities of carbohydrates.

The following appears to have been a case of this kind:—A. B. C—, æt. 59, auctioneer. The

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family history was very good. Father died æt. 97; mother died æt. 78. Habits good. Healthy appearance. The height was 5 feet 9½ inches; the weight 13 st. 8½ lbs. Organs sound. The urine on the first examination, October 19th, contained abundant sugar; on the second examination, December 7th, it was normal.

The private medical attendant reported, under date December 12th, as follows:—"Mr. A. B. C— consulted me soon after he had seen you. An examination of the urine proved negative—no sugar or albumen. A subsequent examination, however, showed traces of sugar by the usual tests (*e. g.* the bismuth and the picric acid tests). I formed the opinion that the sugar present was not due to impending diabetes, but rather to the uric acid diathesis. Physical condition, age, etc., point in this direction. Big stout men actively employed frequently have traces of sugar without any ill effects arising. I put him on a carefully considered diet. A third examination proved the entire absence of both sugar and uric acid." I felt justified in recommending acceptance, with an addition of three years, but the case was not completed.

Another case in which examinations were made over a period of some months will help to illustrate the difficulty of determining the nature of some cases of glycosuria.

M. R—, æt. 41, Board of Trade examiner. The family history was excellent; father, mother, brother, and five sisters were all living and healthy. The personal history was good. The gentleman was muscular, fresh-coloured, and active. His height was 5 feet 7½ inches; his weight 12 stone. Everything was satisfactory except the urine.

Five specimens of urine were examined between March 19th and April 1st, 1897. On every occasion the colour was bright amber, twice unusually so; the reaction was acid; the sp. gr. varied from 1020 to 1034. Four out of the five specimens gave a dense yellow precipitate with Fehling's solution; the fifth gave a precipitate of a green colour. The phenyl-hydrazin test was applied in two specimens, and produced a yellow crystalline precipitate. Another specimen was subjected to the fermentation test, and fermented with a perfectly fresh specimen of yeast. After the first analysis the proposer was re-examined, and no other evidence of departure from perfect health was found. The medical examiner concluded: "Possibly the sugar is only a temporary ingredient of the urine. I advise either refusal or delay for six months."

The gentleman was re-examined in January, 1898; the urine was bright amber-coloured, acid, sp. gr. 1030, and free from albumen. There was a copious red precipitate of cuprous oxide on adding a few drops of the urine to boiling Fehling's solution. A specimen was submitted to the professor of chemistry at E—, who, without hesitation, pronounced it to contain sugar; while the balance was sent to the proponent's medical attendant, who said at once it was saccharine, but remarked it was the only one of several examined that he had found so. But while all the specimens examined by the medical officer to the insurance company were passed in his presence, all those examined by the proponent's own doctor had been sent to him.

Another specimen, examined February 2nd, showed a copious yellow precipitate on adding a few drops to boiling Fehling. This precipitate was slow in coming, and was not complete for three minutes.

The summing up was: Ineligible for life assurance at present, even if it could be proved that the presence of sugar is intermittent and not continuous.

The proponent's medical attendant wrote as follows on January 10th, 1898:—"I have at intervals during the past six months, on six or seven occasions, examined M. R—'s urine. On each occasion I have found the urine normal in every respect. I was specially directed to the possible presence of sugar, but I have never found any. I can find nothing in the history to suggest a suspicion of diabetes." The life was declined formally on February 9th, 1898. Subsequently we learnt that in January, 1900, the life was accepted by another company, with an extra of eight years, the premiums being limited to seventeen annual payments.

I should like next to say a few words about the glycosuria of obesity.

A considerable proportion of over-fat persons occasionally pass sugar in their urine, and it has been estimated that glycosuria occurs in about 33 per cent. of all cases of marked corpulency. The deposit of fat in the tissues is Nature's way of dealing with a greater supply of carbohydrate in

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the food than is required to supply the needs of the tissues. But there is a limit to the powers of storing up fat, and then sugar is passed on into the urine. If these cases are taken in hand in time and appropriately dieted, sugar will probably entirely disappear, and the amount of fat in the body may be reduced to safer limits. In the early stages these are cases of alimentary glycosuria, glycosuria which is intermittent and dependent on the kind and quantity of food; but the over-stout unfortunately often do not seek advice until symptoms of diabetes have supervened, when the condition becomes less amenable to treatment. According to Frerichs, obesity is present in about 15 per cent. of all cases of diabetes.

I look upon the occurrence of glycosuria in a stout person as a danger signal indicating the need of proper diet and regimen. The immediate prognosis is favourable, but on the whole these cases are not good lives. Out of fifty deaths from diabetes which have occurred among the insured at the British Empire Mutual Life Assurance Company, eight were overweight cases. The average age at entry was forty, and the average age at death was fifty-eight, so that they fell short of the expectation of life, which would be about twenty-six years, by some eight years. For the whole series of fifty cases the average age at death, which was sixty-three, was only two years short of the expectation for the average age at entry, which was thirty-six.

Another clinical variety of glycosuria is that associated with gout, which we may call gouty glycosuria. In some gouty subjects sugar is occasionally and intermittently present, usually in small amounts. According to von Noorden, gout in middle life may be followed by glycosuria in later life, or attacks of gout may alternate with the presence of glycosuria, or an attack of gout and glycosuria may co-exist. The following is a not uncommon type:—A patient, middle-aged, accustomed to live well, consults you for chronic gout which perhaps threatens to produce deformity of his hands. He has no symptoms of diabetes, but you find on examination that his urine contains sugar in considerable abundance. With attention to diet and exercise, and treatment with alkalies, the glycosuria entirely disappears, and with this there is much improvement in the general health. If the man lives carefully, he may be little troubled with gout and be free from glycosuria over a long course of years.

I remember a good many years ago being called in the middle of the night to see a City alderman, a gouty subject, who had been attacked with gastro-intestinal disturbance. He had been specially alarmed because on getting out of bed he had turned giddy, had fallen on the floor, and with difficulty had got back to bed. On examining the urine I found it contained much sugar, and I was inclined to take a more unfavourable view on that account than I would do in a similar case now. The patient soon recovered from the attack, and the presence of sugar proved to be quite temporary. Sir Lauder Brunton mentions the case of a lady who died at the age of eighty-eight, and was known to have had gouty glycosuria for thirty or forty years. I think Sir William Roberts very well summed up the salient features of gouty glycosuria when he said—"It rarely proves severe, it is as a rule unaccompanied by thirst and diuresis, and is efficiently controlled by moderate dietetic restrictions."

Dr. WILLIAM EWART: In my experience the most hopeful treatment of diabetes in the young is by hygiene rather than by drugs. A question was raised to-night as to the value of opium. I have known the days when it was the fashion to give opium, and when very great importance was attached to its administration. I have treated cases after that plan, but I find on looking back that it was not a satisfactory method, and that I did not obtain from it permanently good results. It seems to me that a more rational treatment is to raise the general health of the patient and to leave opium alone, and all sedative treatment which tends to retard metabolism. The only chance for the young diabetic is to supply him with that energy, the lack of which is hurrying him to the grave. More food and a better assimilation are needed, and we should endeavour with the help of a comprehensive hygiene to enable the patient to return to a more mixed diet. Of late years I have had good results from this method of treatment. I have been less exacting as regards the entire disappearance of the sugar, and I have been content to allow patients to gain strength and the power of enjoyment of life at the cost of their still passing a little sugar in the urine. A case in point was that of a young man who had become so much reduced that he was incapable of

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any exertion, and who regained his complexion, his weight, and his strength so as to be able to play games and to enjoy them. That patient was better off, in spite of a small amount of sugar still being passed, than if he had been plied with sedatives and submitted to restricted diet. His treatment had been chiefly tonic and hygienic, and directed to improving the muscles, the respiration, the condition of the blood, and the health generally. Others may have adopted this method, but for my part I did not formerly carry it out as I now do. Some cases are obviously too far advanced to admit of their being treated in this way, but there are many which, if taken sufficiently early, would yield good results by being given a progressive diet in combination with the methods of hygiene to which I have referred. I have found an exclusive milk diet extremely valuable as a first instalment in this method of treatment. In a bad case now under my care, that of a girl of eighteen, marked improvement occurred as soon as the diet was reduced to milk only; and it was obvious that the lactose contained in the milk was doing good, as the excretion of sugar dropped whilst the amount of the urine decreased.

Dr. C. C. GIBBES: Nearly every organ of the body has been referred to in this discussion, the heart being one of the exceptions; presumably, therefore, no positive information can be derived from that organ. I am of opinion, however, that some negative evidence can be obtained when it is affected by certain forms of disease. We find it stated in text-books that when chronic venous congestion is present to a marked degree, sugar may be found in the urine. This, however, is an exceedingly indefinite statement, for we are not told the quantity of the sugar, or whether albumen is a necessary adjunct. On looking over my case-books and taking 200 consecutive cases of valvular disease in which chronic venous congestion has been a marked feature, I find that sugar has only been present in one instance, that is in sufficient quantity to respond to the Fehling reaction. Albumen was present in 171 of these cases. In the case in question the sugar was not persistent; its presence resulted from alcoholic excess.

I do not consider that these 200 cases are of any value from a statistical point of view, but they certainly show that sugar is a rare rather than a common accompaniment of chronic venous congestion. The point that I would deduce from this is as follows:—In chronic venous congestion we have a defective metabolism, an excess of CO_2 in the blood, and an absence of sugar; whereas in glycosuria the CO_2 is greatly diminished in quantity, as Dr. Hale White has pointed out.

I would therefore suggest that this aspect of the subject might be worth consideration.

Dr. HALE WHITE: Gentlemen,—In the first place it is my duty to thank you for the extremely lenient way in which you have received my imperfect opening remarks. Most of the speakers have confined themselves solely to the question of glycosuria. No doubt the exigencies of time made this necessary, but I think it is a pity, for in the first place our President told us that the subject of debate included diabetes generally, and secondly, it always occurs to me that we are more likely to progress in our knowledge of diabetes if we try to link together the different phenomena of it rather than to study one exclusively. It cannot be too strongly insisted that glycosuria is only a symptom, and probably not the most important symptom, of diabetes; for there is no reason for thinking that death is ever directly due to glycosuria, while it certainly is often due to another symptom—namely, coma. Glycosuria—or, to be more accurate, dextrosuria—is a symptom of diabetes, as albuminuria is of Bright's disease; but both occur in other conditions than diabetes and Bright's disease respectively, and a patient with diabetes may be desperately ill when he has but little sugar in his urine, and so may a patient with Bright's disease be near his end with but little albumen. In both diseases the most important thing is to understand the formation of the coma-producing poison. Just think that in diabetes we know that metabolism is perverted in at least six ways: there is the failure to use sugar, there is its production from proteid, there is the effect that ingested sugar has in exciting sugar-formation, there is the formation of oxybutyric acid and its allies, there is probably separate from the last the low tension of carbondioxide in the tissues, and there is the formation of the so-called fatty blood. I think we are most likely to progress if we study these together rather than separately.

Believing as I do, and as I pointed out in my introductory remarks, in the fundamental difference between alimentary and composite diabetes, I am

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naturally a follower of Dr. Pavy in his separation of the two diseases. His remarks raise the question whether it is necessary, for the case to fall in the composite category, that some of the excreted sugar should come from decomposition of proteid. No doubt ultimately in a composite case some of the sugar has this origin, but, on the other hand, I think most would be agreed that, for many years, in a slowly progressing composite case, the sugar in the urine is probably derived entirely from the failure of the body to oxidise it. If I understand Dr. Pavy correctly, anyone may be rendered glycosuric by taking enough carbohydrate, but if that is so the amount in the food necessary to produce sugar in the urine must, in some people at least, be very large; for in the patient of mine to whom I referred as suffering from severe traumatic diabetes insipidus, very large amounts of sugar were given by the mouth, and yet more appeared in the urine, although one would have thought in such a case there was an exceptional opportunity for the sugar to be hurried out of the body before it was used.

Dr. Pavy's remarks as to the difficulty of dealing with these cases in which sugar is only occasionally present will, I am sure, appeal to us all. As he says, it is most remarkable to observe how sometimes there may be a large amount of sugar in the urine, and within two hours none at all. The great difficulty with these cases is to decide whether they are the early stage of genuine composite diabetes or only alimentary glycosuria. If there is no ferric chloride reaction, time only can show. Also we can, I am sure, all bring to mind cases supporting Dr. Pavy in his statement that the presence of but little sugar does not necessarily mean that the case is not genuine composite diabetes; the patient may be suffering severely from this disease with an intense ferric chloride reaction and but slight glycosuria. I was glad that Dr. Pavy referred to pertussal glycosuria, for, although it has been fully described, its presence is often forgotten. His case of lager-beer glycosuria was most interesting, especially as the specific gravity of the urine was only 1008. This naturally makes us wish to know precisely what the copper-reducing body in the urine was.

Dr. Saundby referred to alcohol, and suggested that it was a cause of diabetes. In connection with this we must remember that some of the instances of glycosuria after excessive consumption of alcohol are very likely examples of alimentary glycosuria rather than genuine diabetes, and are probably due to a premature wearing out of the tissues. Many of the cases which in Germany follow excessive beer-drinking belong to this class. Then, too, many alcoholic drinks contain much sugar. All alcoholic drinks contain other substances than alcohol, and there is often too great a tendency to ascribe disease to alcohol when all we know is that it follows the ingestion of alcoholic drinks. As far as I know, there is no proof that absolute alcohol can produce genuine composite diabetes, and also there is no proof that unsweetened alcohol in moderate quantities is harmful to diabetics. Until this is shown to be the case it will be well often to allow alcohol to diabetics, for their carbohydrate-free diet is short of energy, and an ounce of brandy yields about 90 calories. Lævulose has been deprecated because of its expense, but if a diabetic can take two ounces of lævulose he is gaining 250 calories, and the cost of this amount of lævulose is just under a shilling. The cost of four ounces of brandy is also well under a shilling, and is worth 360 calories, so that a diabetic can, by spending about one and ninepence in brandy and lævulose, obtain 610 calories; for a working man this is, of course, extravagant, but it is hardly so for the upper and middle classes. Dr. Saundby alluded to the respective value of potatoes and toast for diabetics, but it must be remembered that bread contains 50 per cent. carbohydrate and 40 per cent. water. The effect of toasting is to drive off the water, so that toast is little but carbohydrate, while potatoes only contain about 18 per cent. of starch. Before it can be accepted that there is any special value in potatoes for a diabetic, comparison must be made not between toast and potatoes, but between equal weights of the carbohydrate in toast and that in potatoes.

The chief part of Dr. Bradford's remarks was directed to pointing out that probably different cases of diabetes owned a different morbid anatomy and a different pathology. With that I think all will agree. The history of medicine shows that the more diseases are studied, the better able we become to see distinctions between what were regarded as the same. Whether Dr. Bradford's suggested distinction will be those ultimately

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adopted time only can show. His mention of glycosuria due to excessive muscular fatigue was of special interest when we remember that Bunge uses the fact that diabetes is often seen in those who are sedentary as an argument that some cases of diabetes are dependent upon abnormal processes in muscle.

Every one will, I am sure, agree with Dr. Hutchison that clinically one of the most important questions is the distinction between diabetic and non-diabetic glycosuria. The distinction is not of essential importance when we are considering treatment, for it is allowed that in all cases, whether examples of diabetes or only examples of alimentary glycosuria, the object of treatment is the same—namely, to diet the patient so that sugar will disappear from the urine,—but the distinction would be, if we could make it with certainty, of essential importance from the point of view of prognosis. Unfortunately we can rarely, if ever, be certain whether a patient who is passing sugar in his urine is only suffering from alimentary glycosuria, or whether he is suffering from the early stage of composite diabetes, if the urine contains no oxybutyric acid or its derivatives, and the sugar is quickly controlled by diet. The proper treatment of such a case is of the greatest importance; the urine must be examined from time to time, especially after an ordinary meal, and directly it is found that such a meal leads to glycosuria the patient must again be dieted. But if the patient should refuse all treatment the distinction between alimentary glycosuria, or, as some call it, alimentary diabetes, and genuine diabetes, or, as Dr. Pavy calls it, composite diabetes, would usually soon show itself; for, as Dr. Hutchison urges, the failure to utilise sugar is, in the case of genuine or composite diabetes, progressive (and, indeed, often rapidly progressive if no treatment is adopted), but this is not so in the alimentary form, or at any rate the failure progresses very slowly. But a distinction based upon rate of progress, although of great clinical value, is of little pathological value, and I would suggest that one pathological distinction between the two conditions lies in the fact that in genuine diabetes, if untreated, the output of sugar in the urine becomes greater than can be accounted for by that in the food; and, indeed, often there is sugar in the urine when there is none in the food, showing that the sugar has been formed from proteids; but in alimentary diabetes all the sugar in the urine is derived from the carbohydrates in the diet. Probably a further distinction lies in the fact that the genuine diabetic sooner or later excretes oxybutyric acid or its derivatives in his urine in large quantities and constantly, while this phenomenon is not a characteristic of alimentary glycosuria; and lastly, the genuine diabetic may at any time become comatose, but the person suffering from alimentary glycosuria has not the tendency to diabetic coma. We have, I am sure, like Dr. Hutchison, met with patients suffering from genuine or composite diabetes who have ascribed their illness to an injury of the nervous system; but I doubt whether much attention ought to be attached to this unless a number of cases in which the nervous lesion is found after death are published. It will be remembered that this subject was thrashed out at the Pathological Society of London in 1883, and it was shown that the appearances in the central nervous system which had been thought to be characteristic of diabetes were either normal or due to faulty methods of preparation, and it was further shown that there was no special liability of inmates of asylums to diabetes. That injuries of the central nervous system will produce transitory glycosuria is, I think, quite undoubted; but that nervous injury will cause composite diabetes is, I think, very doubtful.

With regard to hyperglycæmia as a cause of some of the symptoms, there is no doubt that some—for example, polyuria and pruritus—are due to it, and very likely some others are due to it. My only contention was that many are set down to hyperglycæmia upon insufficient evidence; they may or may not be due to it, but the case is certainly not proven. For example, the cases of persons who have cellulitis and do not improve till the sugar has been removed from the blood have been cited. But the very means adopted to remove the sugar leads to an increase of weight, a general improvement of health, and, if the patient has diabetes, to a diminution of oxybutyric acid in the urine, and for all we know to other changes also. Who can say to which alteration in the patient the healing of the cellulitis is due? If anyone maintains it is due to the disappearance of the sugar, it lies with him firstly to explain why all people with hyperglycæmia do not get cellulitis, and secondly to induce cellulitis by the experimental induction of hyper-

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glycæmia. I feel sure the Society will agree with me that it is better to be content with saying that certain symptoms are perhaps connected with hyperglycæmia than to assert, as some authors do, that they are due to it. We shall all agree with Dr. Hutchison that the strongest reason for withholding carbohydrates is that the patient improves if this is done, and further, after a period of abstinence from carbohydrates he can utilise them much better. Also we should agree with him that in some cases even a restriction of proteids was necessary, but these extremely severe cases are very difficult to treat. I quite agree with Dr. Hutchison as to the advantage of the casein breads, and also that if a patient is on a carbohydrate-free diet the addition of even milk to the diet may lead to an increase of sugar in the urine. In some cases, too, sugar-free milk, as it has been called, and which was suggested by Williamson, is of advantage; but, as Dr. Hutchison himself remarked, on the other hand, often patients are too strictly dieted, and they appear to derive benefit from a small amount of carbohydrate, and it must be remembered that in the opinion of many too sudden strict dieting leads to coma. If, in any case, it is felt that some carbohydrate should be allowed, I think milk is useful, and in actual practice I think it will be found that the cases in which a little milk may be given with advantage are not few, although no doubt in some the lactose in it is harmful. Each case must be judged on its own merits. What I have said already will show how greatly I am in accord with Dr. Hutchison in what he says about the value of alcohol and fat.

I must confess I am surprised there can be any doubt that in some cases, and indeed in many, disease of the pancreas plays an important part. Experimental evidence alone is strong on this point, but apart from this I have made many post-mortem examinations in which a diseased pancreas has been associated with diabetes, and there is no doubt as to the accuracy of the figures from Guy's Hospital, which I quoted in my opening remarks.

Some speakers have assumed that when sugar in diabetes is derived from proteid it comes from the proteid in the food, and no doubt this is usually so, but I would ask whether sometimes it may not come from tissue proteid. The perversions of metabolism in diabetes are so many that this could hardly surprise us. If in any cases sugar has this origin, probably it is in those in which the ingestion of a little sugar when a person is on carbohydrate-free diet leads to the excretion of a far greater amount of sugar than the carbohydrate in the food will explain. While on the subject of the excreted sugar I should like to plead that if in any case restriction of diet does not lead to disappearance of the sugar, small amounts of carbohydrate may be allowed, provided always that they do not lead to any increase in the output of sugar. A diabetic must have food, and if we were to diet him strictly according to theoretical ideas we should give him none, for carbohydrates are bad for him; some excreted sugar may be derived from proteids, and many believe that the poison which produces coma is derived from proteids, while the more recent researches go to show that it is derived from fat.

In spite of frequent failures I think we should try sodium carbonate or bicarbonate for the coma; no other treatment is known, and I certainly think I have occasionally seen good result. Opium will in some cases lead to a diminished output of sugar, and it is a remarkable fact that it is difficult to make sufferers from diabetes—in spite of their tendency to coma—drowsy from opium. Codein, too, will lead to a diminution of the sugar in the urine. It seems likely that the fact that sometimes one drug is useful and sometimes another is due to the fact that we have in reality more than one disease comprehended under the term diabetes.

Dr. Hector Mackenzie, I was glad to hear, agreed with me that the copper-reducing test is unsatisfactory, for, as he said, any concentrated urine will give a doubtful reaction if it is boiled long enough. As he said, the temporary cases are very difficult, for in their early stages they appear to be undoubtedly instances of alimentary glycosuria, and then later they show themselves as true diabetes. We should all agree with him as to the great danger of sugar in obese persons, but even here exceptions are often met with. Our President doubted whether the frequency of sugar in venous congestion was not over-estimated. I think it is, and my experience quite agrees with his that glycosuria is not found oftener than once in about two hundred cases of heart disease.

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THE SIGNIFICANCE OF ACUTE SEPTIC PERITONITIS.

By A. MARMADUKE SHEILD, M.B., F.R.C.S.,
Surgeon to St. George's Hospital.

ACUTE peritonitis should now hardly be looked upon as a disease in itself. In the vast majority of cases it is the consequence of some formidable direct infection which has produced it. Excluding the causation of acute peritonitis by causes from without, the nature and existence of which are usually obvious and clear, we may clinically look upon the malady as due to a septic infection of the membrane from some cause within the abdomen. This cause we have, if possible, to discover, interpret, and promptly treat by surgical aid. Upon the correct appreciation of this principle may often hang a human life. Bullet-wounds, stabs, or lacerations of the peritoneum by the claws or horns of animals are sufficiently obvious. It is well here to draw the attention of the practitioner to two especial localities where septic wounds of the peritoneum are peculiarly obscure and concealed from view. The one obscure situation is through the vagina or uterus; the other is by the rectum. By the former routes the unskilled efforts of the criminal abortionist may produce a septic and fatal peritonitis of a most mysterious type, readily misunderstood by the unwary. An instance is as follows:

Septic Peritonitis from Criminal Abortion.—Many years ago, when house surgeon to the Cambridge Hospital, I saw the case of a young girl æt. 18, who was suddenly smitten with acute peritonitis of a very severe type. In these days I feel sure that the case would have been attributed to perforation of the appendix. The symptoms were attributed to "cold checking menstruation." She was treated by opium and leeches, as was the custom in those days. An abscess formed and "pointed" in the vagina, where it was opened. For weeks she was

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desperately ill, and seemed certain to die. She became alarmed and made a "confession." After a long and desperate illness she recovered with her life, but she was terribly damaged. The pelvic viscera were involved in a mass of adhesions, and both hips were flexed, contracted, and partly ankylosed. No one had the least suspicion of the true nature of her case until she made the "confession." As a result a man and woman were arrested, and subsequently convicted and sentenced to a long term of penal servitude.

So, too, the well-directed efforts of the obstetrician may cause unintentional but fatal infection of the peritoneum. Thus operations upon the cervix uteri, or penetrations of the uterus by the "sound," or especially the curette, have in the best hands caused peritonitis. Such unfortunate cases are in the unpublished experience of most large hospitals, and may occur to anyone in gynæcological practice. If strict asepsis be practised, such lesions do not produce the fatal septic peritonitis which I have personally seen excited by the passage of the sound before the days of Listerism.

Peritonitis from the Use of the Uterine Sound.—Early in the "eighties" I assisted in the treatment of a case of this nature. A young married lady received an obstetrical examination at the hands of one of the best known authorities of the day, and the sound was employed. On her way home in the train she was seized with agonising abdominal pain and collapse, and had to be brought back to London. She was obviously suffering from the worst form of acute septic peritonitis, from which she died. She was seen by most of the leading authorities, including Mr. Thornton, but the practice of opening the abdomen and cleansing it had not then prominently come into vogue. When acting as obstetrical assistant to my hospital about that period, I saw several cases of pelvic cellulitis and suppuration undoubtedly caused by the use of the "sound." Seeing how readily breaches of surface are effected by such agents, we cannot be too cautious in their use, and the employment of the most strict asepsis.

Criminal wounds by the rectum are not unknown to the medical jurist. An enema syringe has caused a perforation of the gut, and so has a "lever" passed into the bowel with the intention of compressing the iliac vessels. In strictured or diseased conditions of the bowel a fatal septic peritonitis may be excited with terrible facility. I have known the mere passage of a finger through a strictured rectum cause a crack in the coats of the bowel, and the use of bougies in such cases is fraught with great danger. All forms of dilators of rectal constrictions are open to the same grave objections—namely, the ready laceration of a diseased bowel and fatal infection of the peritoneum. I commend these remarks to the careful consideration of all practitioners. Nothing is so serious and embarrassing as the production of acute septic peritonitis in a patient by such apparently harmless procedures. In the majority of cases where peritonitis is excited by wounds from without the organisms will be streptococci or staphylococci, or both mixed. Such cases are exceptional in civil practice in this country, and one may pass through a long professional life without encountering them. It is far otherwise with peritonitis excited by infection from within. These anxious and responsible cases are in everyone's experience, and form not a small proportion of the fatalities of human life.

In deference to various authors, I must allude to acute peritonitis from such attributed causes as "cold," rheumatism, pyæmia, erysipelas, or as a sequel to the specific fevers. During a hospital experience in abdominal cases of all sorts extending over twenty years I have never encountered one of these instances; and I believe, therefore, they are sufficiently rare to be dismissed from the notice of the practical surgeon or physician. In the acute peritonitis of pyæmia, for example, is there not some intra-peritoneal lesion, thrombotic or actually pyogenic, whereby direct infection of the membrane is produced? Thus in one case of so-called pyæmic peritonitis an abscess of the liver had actually leaked into the peritoneal cavity; in another a suppurating gland was discovered on the brim of the pelvic cavity. In the so-called and very rare gonorrhœal peritonitis, there is in the male, direct extension by the route of the inguinal canal and lymphatics, and in the female by way of the Fallopian tube.

Far otherwise is it with acute peritonitis due to intra-abdominal conditions. To say that the cause is always septic is not far from the truth. Peritonitis excited by non-septic exciting causes is protective merely. Thus extravasations of blood become surrounded by plastic lymph, decolourised, and subsequently absorbed. The surgeon relies upon

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this process to embed his silk ligatures in plastic exudation, and for the same reason he employs the gauze tamponade.

I have examined cases where iodoform gauze and sponges have been left in the abdominal cavity by the inadvertence of operators, and these formidable extraneous substances were surrounded by masses of partly organised tissue.

It is also right to say that the violence and danger of the peritoneal inflammation will be in direct proportion to the septicity of the exciting agent, and probably also to its amount. No complaint, for instance, is more terrible and rapid than the peritonitis which follows the rupture of an appendical abscess into the general abdominal cavity; whereas the peritonitis excited by a small leak in the walls of the stomach is often limited, and, indeed, may result in a localised abscess, and be recovered from, without the prompt surgical measures so essential in the former condition. Thus the *severity of the initial symptoms* may often with justice be looked upon as a most important diagnostic point in estimating *the gravity and extent* of the perforating lesion and the peritonitis likely to ensue.

Healthy secretions, as bile, urine, or gastric juice, would in theory produce a plastic, non-suppurative peritonitis. In practice this is seldom or never the case. The diseased conditions which lead to the extravasations furnish a plentiful supply of septic organisms mixed with the secretions. In "squeezes" or crushes of the belly, where viscera are wounded without external wound, the coats of the intestine, if not actually ruptured, are often bruised or deprived of their blood-supply from injury to the mesentery. Such conditions afford means by which bacteria can penetrate the walls of the intestinal tube, leading to rapid decomposition of fluid collections in the immediate vicinity. Acute peritonitis coming on after a blow on the belly is a symptom which usually justly leads to the belief that some internal viscus has been injured. As the organisms which produce peritonitis from without are frequently streptococci or staphylococci, so the organism which inflames the peritoneum from within is principally the well-known *Bacillus coli*. At the same time other organisms are found, and the bacterial examination of the products of a septic peritonitis is usually highly complex. The question of the amount of dosage is of great importance in connection with the history of the *Bacillus coli*. The symptoms are proportional to the quantity of the septic agent introduced.

The protective action of peritonitis must be clearly recognised, and the plastic exudations which surround extravasated blood, gastric contents, and pus are familiar to pathologists, and are essential aids to the surgeon. The presence of protective adhesions in peritonitis was recognised by the older authors, and especially by Watson.* The encysting action of plastic exudations is, of necessity, limited and localised, and thus the phenomenon of a distinct lump or mass within the abdominal cavity in cases of peritonitis is, generally speaking, a hopeful sign as regards the saving of life, since it signifies that the inflammation, however dangerous, tends to be limited in extent, and may be dealt with on the principles of an abscess. This will be again alluded to in speaking of appendical abscess.

The vast majority of cases of acute septic peritonitis originate in infection from the alimentary canal. In practice the thoughts of the surgeon should at once turn to the appendix as the cause of the evil. *In children and young adults a sudden acute and dangerous peritonitis is far more likely to be due to perforation of the appendix than anything else.* Were this dictum more widely known and appreciated many lives would be saved which are now lost.

Next in frequency come the perforations of the stomach and duodenum. These dangerous lesions form a significant proportion of causes of acute peritonitis in adult life. In women are superadded lesions and diseases which occur about the pelvic generative organs. Thus the rupture of an ovarian abscess or suppurating cyst, the sudden extension of infection from a suppurating tube, are familiar examples of causes of peritonitis of pelvic origin, often difficult and dubious enough in actual practice to diagnose and verify. A large number of cases must also be mentioned which are more exceptional, and which may never be met with in practice. The perforation of typhoid fever forms a class in itself. Suppurations and perforations of the gall-bladder, the rupture of hydatid cysts, the extravasation of hepatic, spinal, or glandular

* 'Principles and Practice of Physic,' vol. ii, p. 378.

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abscesses into the abdominal cavity, are all exceptional causes of peritonitis which I have met with in practice in the last twenty years.

I have also encountered several cases of acute peritonitis in connection with cancer of the large intestine, the colon, or upper part of the rectum, sudden leakage of virulently septic material producing a violent septic peritonitis, the nature of which was only clear after exploration by operation. It is very noteworthy that in these cases the existence of previous cancerous disease of the bowel was quite unsuspected, the patients having merely suffered from vague disturbances of the digestive tract. This is a good example of such an illness:

A Case of Sudden Perforation of a Cancerous Colon, with Acute Septic Peritonitis as a Consequence; Symptoms of Intestinal Obstruction.—A stout man æt. 64 was admitted at midnight into St. George's Hospital on October 19th, 1900. His belly was distended, tympanitic, and tender. His pulse 124, "small and hard." The temperature was 100°. The tongue was foul, and the patient had been vomiting for three days, at intervals. Absolute constipation existed. The face was pinched and anxious, and the patient evidently desperately ill. There was nothing to be felt on rectal examination; extreme tenderness was elicited. He had had large doses of powerful purgatives. The diagnosis of chronic intestinal obstruction was made. The patient being carefully anæsthetised with A.C.E. mixture, the left iliac incision was made, and a cancerous growth of the sigmoid was at once found. The belly was full of thin, stinking pus, the intestines red, injected, covered with flakes of lymph. The cause of this was not far to seek. A ragged perforation existed in the cancerous mass, and feculent material could be seen issuing therefrom. The diseased bowel was drawn out of the wound and attached, and the peritoneum cleansed; but, as might be expected in so desperate a case, the patient soon succumbed. It is very significant that the only premonitory symptoms in this case were vague abdominal pains and a tendency to constipation, for which purgative drugs had been employed. The abdominal pain came on suddenly when the man was driving his van four days previously.

This case is only one of several I have seen, and sufficiently demonstrates that cancer of the large intestine may exist unsuspected, and sudden perforation and septic peritonitis may occur from it, the cases being difficult and misleading in a high degree. In other such cases a gaseous or fæcal abscess may form and "point," or be opened through the abdominal wall. Here the protective inflammation of the peritoneum is sufficient to encapsule the pus.

Acute peritonitis from perforation of the intestine by foreign bodies belongs to the "unusual" and "curious" category. Such offending materials as pins and pieces of bone more often cause localised abscesses than general acute peritonitis. Cases are, however, on record where jugglers and mountebanks have paid with their lives for their temerity in swallowing knives, nails, pieces of glass, and the like; and a similar fate has befallen lunatics, who may swallow dangerous substances. A mysterious peritonitis in an inhabitant of an asylum may be explained in this way. Such cases are not likely to come under the notice of the practitioner, and I only allude to them for the purpose of completeness. My notes only contain two instances of peritonitis of this class. The one was due to perforation of the appendix by a pin; the other was caused by a portion of jagged bone which worked its way through the descending colon. In both of these cases the peritonitis was localised. The use of the X rays may be of great value in mysterious localised peritonitis in a person of unsound mind, and the employment of this aid to diagnosis should never be neglected.

A proper knowledge and appreciation of the initial symptoms of perforative peritonitis is so important to the practitioner that attention cannot be too forcibly directed to a matter fraught with such momentous issues. The peritoneum and intestines being widely concerned in the distribution of the sympathetic nervous system, any sudden injury is associated with two phenomena of grave import; the one is pain, the other shock and collapse. The immediate condition of a patient suffering from a large perforation of the stomach, for instance, differs little from a man or woman who has been shot in the belly by a revolver. The face is ghastly white and anxious, sweat bedews the forehead, the extremities are cold, and the pulse can hardly be felt at the wrist. I have known instances of patients dying in this condition from perforation of the stomach, and such cases may not survive for the symptoms of subsequent peritonitis

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to show themselves. The severity of the pain is equally marked in bad perforative lesions. Thus it is described as "cutting," "stabbing," burning,—indeed, so severe as to be compared to imaginary agonies, which the patient has never actually experienced. The sufferer is "doubled up," and may cry out, groan, or shriek. *Such significant and terrible pain is peculiarly often experienced in the umbilical region in cases of sudden rupture or perforation of a diseased appendix.* And here we may formulate an obvious, if often neglected rule, that such symptoms as these indicate a formidable injury to the peritoneum, which is not likely to be recovered from without prompt surgical aid.

The situation of the pain is only a rough guide to the situation of the lesion. Often, when an abscess or a viscus perforates, the septic fluid is ejected against the peritoneum or into one of its numerous pouches, at some little distance from the actual seat of mischief. The pain and tenderness will be worst at the situation where the inflammation begins to originate.

Then there are the errors which patients frequently make in cases of "referred" nerve pains, originating in areas from whence the mind has little experience of real sensations. Thus in acute appendicitis the pain is often referred to the umbilical region, and in perforations of the duodenum the pains may be referred to the "pit of the stomach," the umbilical region, or even to the left hypochondrium. There is a case of an elderly man recently operated upon for acute abdominal symptoms; his pain was referred to the region of the spleen, but the actual lesion was a perforation of small size near the pyloric end of the stomach. Following upon the initial symptoms rapidly supervene others of vital importance, due to inflammation of the peritoneum and rapid absorption into the circulation of the deadly toxins produced by organisms in the great serous sac.

Local tenderness to pressure with the hand is universal, and this is associated with sudden rigidity of the abdominal walls over the damaged area. Both these signs are invaluable, as assisting the anxious practitioner in distinguishing between perforation and the "colicky" pains attending upon the ingestion of unsuitable or irritant food. The pulse now assumes the well-known quick and hard, or "thready" character, so often found in inflammations of serous membranes associated with extreme sympathetic nerve distribution. Rapid distension of the belly ensues, with absence of respiratory movements. And now, as the inflammatory process has spread to the walls of the intestine, the gut becomes red, covered with lymph, and distended with gas. This is marked clinically by inability to pass flatus *per anum*, by hiccough and belching of wind, and subsequently by vomiting of bilious material.

Vomiting is frequently an exceedingly early and important sign in perforative peritonitis. In sudden perforations of the appendix it may come on almost immediately, and in perforating gastric ulcer the symptom may be so prominent as to lead to the erroneous diagnosis of irritant poisoning. The symptom is, however, a variable one in the early history of the affections we are considering. In the later stages, however, definite intestinal obstruction is present, or perhaps a better expression would be definite paralysis of the intestinal walls with inability to expel the contents of the intestines.

This great clinical fact needs accurate appreciation. The wrong diagnosis of acute intestinal obstruction from "a band" or internal hernia is too readily made in these cases, and the early treatment of perforative lesions by lethal doses of purgative drugs is due to a confusion in the mind of the practitioner as to whether the symptoms are not due to some accidental obstruction. *A clear knowledge that peritonitis causes obstruction may save this disastrous practice.* Nothing is worse in a case of perforated appendix or a duodenal ulcer than a full dose of calomel or a measure of castor oil. Yet how frequently do we see this fatal error committed, and the chances of the patient made infinitely worse! Peristalsis excited when there is a hole in the bowel merely leads to the ejection of fæcal material, and aggravates the dreadful evils already present.

The later stages of perforative peritonitis are well known enough. They are those of advanced septic intoxication and rapid collapse of the vital powers. The pinched face and deep hollows under the eyes; the tumid belly, too often marked by inefficient leech-bites and discoloured by "turpentine stupes;" the pulse weak and thready, hardly to be counted at the wrist for speed; the cold fingers and toes,—all form a picture formerly very familiar, and even now too often seen in private practice. Death is imminent, and the chances of averting it by

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operation are slender indeed. The mind of the patient may be absolutely clear, and often immediately before death there is a curious cessation of pain, which makes the already moribund patient declare himself suddenly much better. I have more than once known the medical attendant to share in this erroneous opinion, and to give a favourable prognosis to the delighted relatives, which was rudely dispelled by the death of the patient only a few hours after!

The time in hours or days that a case of perforative peritonitis may take to run its fatal course is very variable, and varies according to the septicity of the exciting agent and the resisting powers of the individual against the poison. The peritoneum is an enormously absorbent sac, and when distended with virulent stinking pus the quantity of toxins absorbed will soon destroy life. A strong boy of sixteen, for instance, will struggle for days against a septic process which would destroy an adult with naturally weak cardiac action in twenty-four hours. This consideration is a very vital one for treatment, and a surgeon may take operative measures in a young and strong individual which would be perfectly hopeless in less vigorous a subject. Anyone who has had much experience of these cases can recall the instances of boys whose abdominal cavities were full of pus, whose intestines were red and distended, and yet who have recovered under cleansing the cavity and full doses of saline purgatives. Such happy results are too exceptional; still they occur, and youth is a most important factor in the consideration of possible recovery. The temperature in acute peritonitis is a very variable and too often a deceptive factor in the diagnosis. In young and plethoric individuals fever may be marked, especially at the onset of the attack. With absorption of septic products and toxæmia the temperature may fall rather than rise, and the absence of fever may really be explained by a serious diminution of the vitality and resistent forces of the individual. Again, it is a most instructive and important fact that localised abscesses, especially in the right iliac fossa, may exist with but little concomitant fever. *The absence of a febrile temperature is no proof that an abscess in this situation does not exist.* I am able to affirm this positively from abundant personal observation, and it is a clinical consideration too little appreciated. The medical attendant relying upon his thermometer can hardly credit that four or five ounces of stinking pus may be present in the iliac fossa. Indeed, an incision is often needful both to convince the practitioner and to evacuate the pus.

No account of the phenomena of acute peritonitis would be complete without a reference to the remarkable power of morphia over the symptoms. The agonising pain is at once lulled, and the pathological road to certain death may be completely smoothed by the deceptive sedative influence of this drug. It is indeed difficult to believe that the grateful and drowsy patient who now declares his relief and improvement is the same man who was "doubled up" in abdominal agony a few hours before. The local tenderness even is less, and the one symptom which may make it clear to the cautious and observant practitioner that the true source of evil remains unrelieved, is the terribly rapid and hard pulse.

In private practice, when the early agony of perforative peritonitis is very great, the attendant is almost driven by the importunities of the patient and his friends to do "something." Let him remember to administer the smallest possible dose of morphia. I have never seen necessity for more than a sixth of a grain hypodermically in these distressing circumstances. Even this quantity masks serious symptoms, and is very apt to render the need for prompt surgery less urgent in the eyes of all concerned.

The treatment of acute peritonitis by what are termed medical measures is open to the great objection that the medical attendant must feel uncertain as to whether or not a hopeless lesion remains undetected, which must render his best efforts nugatory. The decision has to be made promptly, for nothing is more distressing and useless than to invoke the aid of surgery to a distended moribund patient, who, to obtain any chance of life, should have been operated upon days before. If medical measures have failed in these cases, surgery indeed has a poor chance. Accordingly I feel sure that few indeed of the forthcoming generation of medical men will have the hardihood to treat by medical measures a violent peritonitis coming on suddenly, with the pain and sickness and other symptoms of grave import I have attempted to depict.

In those instances when patients or relatives

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refuse operative treatment, what is to be done? Such experiences are not uncommon among the votaries of homœopathy or Christian Science, and also among the ignorant in rural districts. Now the experience of anyone who has been long in practice teaches that acute septic peritonitis may in exceptional instances be recovered from without surgical aid. In such cases the patients are usually young and vigorous, and the initial infection slight. I have on several occasions known severe peritonitis having its origin in the neighbourhood of the pylorus get spontaneously well. In such cases I have always thought that some small lesion of the duodenum was present, but of course one could not be sure. Again, it is difficult if not impossible for a medical man single-handed, living perhaps miles from aid or from the assistance of a competent anæsthetist, to undertake an exploration, let us say, for a supposed duodenal ulcer. This aspect of the question has been much ignored by writers, who appear to little know or appreciate the difficulties which beset the path of the practitioner dwelling in remote districts.

Small doses of morphia mixed with atropine, the emptying of the large intestine by enemata, and the support of the strength by rectal alimentation, are the main factors upon which the practitioner must rely. Leeches and turpentine applied to the abdominal wall are only of slight benefit, and doses of calomel by the mouth are only permissible in those cases when no perforation of the intestine occurs. In this haphazard mode of treatment the practitioner must, to speak plainly, rely largely on luck. If the lesion and source of infection be slight, and the patient young and vigorous, the powers of Nature may be sufficient for recovery. If the infection be severe, and the patient obese or feeble, death will ensue despite all the resources of the pharmacopœia. It is in cases treated after this fashion that the aid of surgery is invoked too late, and operations fall into discredit.

There are few regions in Great Britain where a patient if poor cannot be at once transferred to the nearest hospital, or if well-to-do cannot command the services of a competent operator. The telegraph and express trains cut away the ground from under the feet of those who hesitate to obtain skilled aid.

We are now in a position to formulate a rule of practice, which can, indeed, be open to but few exceptions. It is as follows:—Whenever a patient is smitten with the group of symptoms which I have described, abdominal exploration by a skilled operator gives the best chance for life, to repair the lesion, to cleanse the peritoneum, and stay the progress of the fatal septic inflammation. That all lives will be saved by these means is, of course, saying too much. Desperate conditions will be sometimes found beyond the hope of human aid to repair. Often, however, it is the cry of "too late!" The intricate pouches and folds of peritoneum are polluted beyond hope of cleansing. Any period of delay beyond six hours in perforative septic peritonitis means the possible destruction of the patient. Heavy indeed is the responsibility of the practitioner in these cases, and fortunate is he who, in his hospital career, has well studied and appreciated them.

The question may well be asked,—are there any preliminary symptoms by which we may be guided to the cause, in any given case of acute perforative peritonitis? Pathological examination affords us a useful answer to this important query. I have already stated my belief that if a boy or girl under the age of twenty is suddenly smitten with severe abdominal symptoms running on to rapid peritoneal inflammation, the cause is practically always in the appendix, which is commonly enough gangrenous or perforated. In a young girl the sudden rotation of a small ovarian cyst may produce symptoms very like those of perforative peritonitis.

Close investigation may show that the patient has had previous attacks of pain and tenderness about the right iliac region, with "colic" and digestive disturbances. Such a previous history makes the mind incline very strongly to the appendix as the origin of the trouble. *The majority of cases of acute, dangerous appendicitis have had these premonitory symptoms.* This clinical fact is of the utmost importance, and cannot be too strongly insisted upon.

Perforating gastric ulcer is common in anæmic young women who have long suffered from chronic dyspepsia and have swallowed much physic for its relief. A history of this nature will much help us. Yet this lesion may be absolutely sudden, and I have known it occur in women apparently previously in the best of health. In ulcer of the duodenum the patient is usually an adult male. A previous history of pain and indigestion is common,

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but by no means universal. The lesion is usually sudden, and if mysterious acute abdominal symptoms, terminating in peritonitis, originate in the upper zone of the abdominal cavity in an adult male, a duodenal ulcer is the likely cause.

Peritonitis excited by suppuration around or perforation of the gall-bladder is comparatively rare. The patient will be an adult, frequently obese, with a history of a lethargic life, and perhaps previous attacks of biliary colic or transient jaundice. I have never known a case of suppuration and perforation of the gall-bladder to occur in which there has not been such premonitory signs of trouble.

In these cases the portal vein or its tributaries is frequently infected, and high fever with severe rigors is common, leading to the wrong supposition of malarial fever.

Peritonitis of pelvic origin occurring in a young married woman is very suggestive of infection from the neighbourhood of the Fallopian tube. Such cases are generally gonorrhœal in origin, and a purulent vaginal discharge may also be detected. Acute peritonitis from criminal operations upon the uterus is for obvious reasons usually devoid of history, or the patient may actually mislead the medical attendant. The practitioner must remember that such a case may be brought before his notice in the most unsuspected quarters, and in the highest walks of what is termed "fashionable society."

It may be supposed that acute peritonitis would offer symptoms so prominent as not often to be mistaken.

In very obese subjects, the layer of fat renders the phenomenon of local tenderness on pressure hard to be elicited. Rectal and vaginal examination may elicit extreme typical tenderness, and if fluid exudation has taken place it may thus be detected. Rectal examination should never be forgotten under such circumstances. In feeble and elderly persons the acute signs of peritonitis are rapidly masked by symptoms of general poisoning of the individual; and abdominal distension, with a quick "running" pulse, may be the only sign of the serious condition present. Finally it may be recollected that in exceptional cases tuberculous disease of the peritoneum may run a very rapid course, and may even be difficult to distinguish from an appendical infection.

The following rules of practice may be formulated from the previous remarks:

Acute peritonitis is the result of infection, and the lesion which produces it usually needs surgical treatment.

The speading of peritonitis over a larger and larger area, no limiting mass of inflammatory nature being detected, is a serious symptom, and calls for operation.

The treatment of acute peritonitis without operation is generally of the "haphazard" type. Recovery may take place if the infection be slight. Many lives are lost through this practice.

For purposes of a practical diagnosis the abdomen may be divided into two zones—an upper and a lower.

In the upper zone the cause of the acute peritonitis may be in the stomach, duodenum, or gall-bladder; in the lower zone the appendix, or the pelvic generative organs in women.

Anomalous and rare causes of peritonitis, as the rupture of a spinal abscess, or a hydatid cyst, will seldom be correctly diagnosed without exploratory operation.

Multiple Melanotic Sarcoma unsuccessfully treated by X Rays.—J. P. Marsh urges X-ray therapeutists to have their work in large, rapid-growing sarcomata checked up by examination of the blood, especially as regards the leucocytosis. Although the X ray seems to be of value in the ordinary slow-growing epitheliomata of the skin before glandular development has occurred, still the X-ray therapeutist should be willing to apply the same three-year rule to his work as does the general surgeon. Several times during this treatment of cases of recurrent cancer of the breast the writer has felt that, although the local condition has been benefited, metastases in the internal organs occurred more quickly than they used to do under the old methods of treatment. In the case reported, of multiple melanotic sarcoma, the writer had the question of leucocytosis investigated, and the blood examinations showed a steady fall in the number of leucocytes with each treatment. It seemed as if the X-ray treatment was having a direct detrimental effect.—*Medical Record*, April 25th, 1903.

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CLINICAL LECTURES
ON
DISEASES OF CHILDREN.

LECTURE XI.

THE RESPIRATORY DISEASES OF CHILDREN.

Delivered at the London Hospital, November 25th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
Assistant Physician to the Hospital, and to the Hospital for Sick Children, Great Ormond Street, W.C.

GENTLEMEN,—To-day we come to the consideration of the respiratory diseases of childhood. Of course I need hardly remind you that that is a large subject, and so in dealing with it it will be well for us to concentrate our attention chiefly upon those diseases of the respiratory system which are commonest in early life, or peculiar to it; and for the rest, to glance simply at those points in which the respiratory diseases of children differ from the same affections as exhibited in grown-up persons. And in taking this course I must run the risk of sometimes appearing to be rather fragmentary.

I must remind you in the first place of certain peculiarities of the respiratory organs in children to which I directed your attention in the first lecture. You remember the peculiar shape of the infant's chest, how it is circular instead of being oval as it is in the adult; one result of which is that the child's lungs can only expand easily in a vertical direction, and not nearly so much from side to side as do the lungs of adults. It is perhaps in consequence of this that collapse of the lower lobes of the lungs in children is apt to be rather common. I would remind you also of certain changes in shape which the chest may undergo in consequence of disease. These changes are rendered possible by the fact that the wall of the child's chest is soft and its cartilages yielding, the result of which is that they get driven in easily under ordinary atmospheric pressure when there are abnormal conditions present in the lungs. I would remind you of the rickety-shaped chest. You will remember that a falling in takes place at each side along the line of attachment of the ribs to the cartilages; and I would again direct your attention to the pigeon breast, which you must distinguish from the rickety chest, and in which the chest wall comes to a peak or keel, as it were, in front. That condition is commonest in diseases in which there has been a great strain thrown upon the chest for a long time, and in which there has been some consolidation of the lower lobes of the lungs, particularly, perhaps, after whooping-cough. There is a third type of chest which you should be familiar with, and that is the adenoid chest, which occurs in older children. Its chief peculiarity is a sinking in of the xiphisternum and lower costal cartilages. Later I shall show you illustrations of it with the aid of the lantern. Then there are certain peculiarities of the physiology of respiration in children. There is the great ease with which respiration in children becomes irregular, and in which the rhythm becomes altered on the advent of any acute disease, with the appearance of the inverted rhythm which I described to you before. I would remind you also that the breath-sounds are extremely harsh, that puerile breathing is the normal, and that on account of the great conductivity of the child's chest you may get accompaniments produced on the one side heard on the other side, and in consequence of which the breath-sounds are apt to penetrate to the surface of the chest, even although there may be a layer of fluid of some thickness between the lungs and the end of the stethoscope. All these points should be borne in mind when you are studying respiratory disease in young children.

We will now look at the respiratory diseases in detail, and I shall begin with the diseases of the larynx, because in young infants they are very important and often very fatal. You will meet in practice with a group of cases, usually in children between the ages of one and five, in which the most striking symptom is stridulous or "croupy" breathing. These cases are much commoner than you might suppose from your observation here, for but few of them find their way to hospitals. You will find, too, that most of these cases are spoken of by parents as "croup." That, gentlemen, is a very bad term, and one which I would advise you to avoid the use of as far as you can. Few terms in medical nomenclature have been the cause of greater confusion in teaching and reading than this word "croup," for it has been used without discrimination by different writers to designate totally different diseases. What I wish you to grasp quite

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clearly is that croupy breathing means simply laryngeal obstruction, and that such obstruction may be due to one of two distinct pathological processes.

1. To mere catarrh, with or without transient exacerbations of the obstruction from muscular spasm.

2. To membranous exudation in the larynx, which is practically always diphtheritic.

We shall devote our attention chiefly to cases of the first group, for you have opportunity of becoming familiar with diphtheria elsewhere.

One can distinguish two varieties in the catarrhal cases, which, however, are by no means sharply marked off from each other. These are (1) mild cases of laryngeal catarrh in which there occur temporary exacerbations of the obstruction from muscular spasm; (2) more severe cases, in which the swelling of the laryngeal mucous membrane is sufficient to oppose a more or less permanent obstacle to the entrance of air.

It will be convenient to have separate names for these groups, and so, following Goodhart, we may speak of the former as cases of *catarrhal laryngeal spasm*, and reserve for the more severe cases the term *catarrhal laryngitis*.

The history of a case of catarrhal laryngeal spasm is usually this:—A child of about three years old has a slight cold, and, perhaps, a rather metallic cough. He goes to bed, however, as usual, without much being thought of it. He falls asleep, but an hour or two later wakes up in an attack of dyspnœa accompanied by crowing inspiration, and in a state of considerable fright. These symptoms last for from a half to three hours, when they subside almost as quickly as they began, and the child falls asleep again. Next day there is probably still some metallic cough, and the croupy attacks are apt to recur for two or three nights. Such a case is probably never fatal. *Catarrhal laryngitis* also begins with a cold, but the croupy breathing and dyspnœa steadily increase, and there are no intermissions as in the milder variety of the disease. At the same time there may be a considerable degree of fever. Such a case will probably last for a week or longer, and you must also remember that laryngeal catarrh may occur at the outset of other inflammatory diseases, such as measles or, as in a case we had in the ward recently, pneumonia; and you must take care not to overlook the signs of these if they are present. You should always look upon laryngitis in children seriously. It is by no means uncommonly fatal, and I have had the misfortune to see two cases die on the table whilst tracheotomy was being performed.

We come now to the important matter of *diagnosis*. Catarrhal laryngeal spasm is usually easy to recognise. In most of your cases there will be a history of previous attacks, and the sudden onset in the middle of the night is very characteristic. Catarrhal laryngitis in its severe forms is much more difficult to be sure of. Such cases will often cause you great anxiety at the outset, for you may have great difficulty in excluding laryngeal diphtheria. I believe, indeed, that the diagnosis between the two may sometimes be impossible, for the signs of obstruction are the same whether it be due to mere swelling of the mucous membrane or to membranous exudation, and there is nothing definite in the general symptoms to guide you. The only conclusive test would be a laryngoscopic examination, which it is, of course, almost impossible to perform. The chief points to be attended to, however, are these:

1. The more rapidly the symptoms set in, the more likely it is that you are dealing with simple and not membranous inflammation. As Trousseau has said, "simple laryngitis makes a great *fracas*, but diphtheria insidiously instals itself."

2. The voice is more apt to be lost in diphtheria than in laryngitis, and the cough to be less, for the membrane protects the mucous surface from irritation. Diphtheria, in short, is the more *silent* disease of the two.

3. The constitutional disturbance, but not the temperature, is usually greater in diphtheria, and the child looks more *ill*; but this, of course, is an indication which it requires some experience to appreciate.

Enlargement of the glands in the neck is of doubtful value, for although that occurs in diphtheria, yet children who are most predisposed to laryngitis from an unhealthy condition of the throat are apt to have it too. This practical rule you will do well to lay to heart: *if in doubt regard the case as one of diphtheria, and treat it accordingly.*

Let us pass now to *treatment*. In dealing with a case of catarrhal laryngeal spasm you have to

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consider (1) what you should do when summoned during an attack; and (2) what steps you are to take in order to prevent recurrence.

When confronted with a case of catarrhal laryngeal spasm you will remember that, no matter how alarming the symptoms may appear, such cases are never fatal, and so you will keep calm and endeavour to allay the natural anxiety of the friends. Your next step should be to administer an emetic. You will find powdered ipecacuanha the best for your purpose. Give a 10-grain dose and repeat it in a quarter of an hour if necessary. Ipecacuanha wine is often recommended, but it is a much less trustworthy preparation. I have given as much as an ounce of it in teaspoonful doses every quarter of an hour without producing emesis, but if you get a fairly fresh preparation of the powdered root you will rarely fail with it. The next thing to do is to apply hot fomentations over the larynx and to arrange some method of inhalation. The best plan is to rig up a tent over the child's cot and to let the steam from a bronchitis kettle play into it. You may use a little creosote with the kettle if you like, but the great thing is to secure the local internal application of moist heat. These measures will usually be speedily successful, but if you are dealing with a severe case it may be justifiable to administer a whiff of chloroform to allay the spasm. In order to prevent the recurrence of an attack the following night you should continue the administration of small doses of ipecacuanha throughout the next day, and at bedtime administer a dose of antipyrin, one grain for every year of the child's age.

As most of these children are predisposed to their attacks by having enlarged tonsils or adenoids, it is well to attend to the condition of the throat in order to prevent the reappearance of the disease. Cold also should be guarded against, for it is a potent exciting cause. In Scotland attacks of "croup" (as it was called) used to occur with special frequency on Saturday nights, the probable explanation being that nursery floors were usually washed on that afternoon in preparation for Sunday, and the attacks were brought on by children being put to bed in a room which was rather damp.

In the treatment of *acute laryngitis* I would remind you again that if you have the least suspicion that the case may be one of diphtheria it is your duty to give an injection of antitoxin and to take all the precautions usual in dealing with the more serious disease. The rest of your treatment must proceed much on the lines already laid down for catarrhal laryngeal spasm. Inasmuch, however, as the obstruction here is organic and not spasmodic, you will expect less benefit from emetics. Should the case be a very "florid" one, *i. e.* the temperature high and the distress great, the old so-called "antiphlogistic" treatment often gives very gratifying results. It consists in bloodletting either from the jugular vein or by the application of a couple of leeches above the sternum. A full dose of calomel should also be given and antimonial wine administered in five-minim doses every two hours. If the dyspnœa increases you must be prepared to perform intubation or tracheotomy, and it is well to remember that it is safer to operate too early than too late. Of the relative merits of the two procedures it is scarcely my province to speak, but I would only say that I believe nearly all the advantages lie with intubation, and I should greatly like to see its more extended use in this hospital.

Other laryngeal diseases in children about which you may make mistakes are congenital laryngeal stridor and laryngismus stridulus. These are totally different diseases, and I shall have occasion to describe them when I come to speak of the neuroses of children, so I only mention them here for the sake of completeness. There is another condition which you should bear in mind when a child has dyspnœa of laryngeal origin, and that is the possibility that you are dealing with papilloma of the larynx. That occurs not uncommonly in children; more commonly, I think, than in grown-up people, and oftener in boys than in girls. The child is usually about three or four years of age, and the symptoms simulate those of chronic laryngitis, for which papilloma is apt to be mistaken. So if you have a child brought to you with a husky voice and occasional slight dyspnœa, and a history that the symptoms have lasted for weeks or months, you should think of laryngeal papilloma. I think you can make an exact and certain diagnosis only by the use of the laryngoscope, and that is a matter of such difficulty that I advise you to send those cases to specialists; in any case you will require a specialist to operate upon them, because the only successful treatment is removal of the growth with forceps through the mouth, which is a very delicate and difficult procedure.

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ACUTE RHEUMATISM AND SOME OF ITS CONCOMITANTS.*

By W. H. COUPLAND, L.R.C.P.Edin.,
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MR. PRESIDENT AND GENTLEMEN,—The remarks that I am about to make will, I fear, be somewhat desultory, but I think that the subject is one especially suitable for consideration by a clinical section like our own; rheumatism being a complaint that we all have to treat in one of its forms on frequent occasions, as it is a disease of widespread distribution.

I do not purpose speaking of the chronic types of the malady, as I believe that they are of quite different etiology to the complaint generally called acute rheumatism, or rheumatic fever. This condition is interesting from many points of view. Its very frequency makes it of great importance in the eyes of the practitioner; and there is further interest in the great success that the usual treatment often leads to. We talk in a case of acute rheumatism of "curing" the patient, but we are apt to forget the sage maxim of Sir Geo. Humphrey that "nothing gets quite well," and we also often forget that our present methods of procedure in this disease are based on empirical lines, the "*causa causans*" being only as yet uncertain.

Although we are not at present able to tack on the name of any organism to the disease and to fully satisfy the four or five postulates of Koch, without which test modern bacteriology refuses to take any organism into its etiological bosom, yet the last three years have much enlightened us as to the relation of germs to acute rheumatism. The experiments of both English and foreign observers have made it clear that the organism is almost isolated. By the kindness of Dr. Poynton I am able to show you a slide from a culture of an organism which was isolated by himself and Dr. Paine in 1900. You will see that it is a diplococcus, small in size, being only $\frac{1}{2}$ mm. in diameter, and it is certainly one of the organisms producing rheumatic fever. They have isolated it from blood, urine, pericardial fluid; also from the tonsils and from growths on the cardiac valves. The injection of cultures of this diplococcus into rabbits caused pathological lesions akin to those found in man. As Achalme had previously found a larger organism, but a bacillus, its claim to be the sole cause of acute rheumatism is at present *sub judice*.

The older theories of the lactic acid diathesis, or the "nervous" theory of the American, Mitchell, may, I think, be safely laid on one side at the present day; but that the bacterial origin of the disease is not quite settled yet is shown by the definition in the last edition of Quain's 'Dictionary of Medicine.' This, which is framed by Mitchell Bruce, runs as "an acute disease; caused by certain infective, diathetic, or climatic influences." The text-book of Osler, dated 1901, has a definition of a much less non-proven character, viz. "an acute, non-contagious fever dependent on an unknown infective agent." I fear neither of these is very illuminating.

I think the more general idea is that the germ is a saprophytic one, and that it is easily killed on entry into the tissues; the disease being infective but not contagious. Although we accept the bacteriological origin of the attacks, we must not forget that other influences act as predisposing causes, *e. g.* a chill. Monti has, for instance, shown that dogs are much more liable to pneumococcus infection after a cold bath; and Pasteur that fowls, naturally immune to anthrax, will succumb when exposed to cold.

No less an authority than the King's physician, Sir Thomas Barlow, has said that the "difficulty in discussing rheumatism consists in defining what we mean by it;" there is little difficulty in the classical attack in the adult, or in the adolescent from fifteen to twenty years, as they are very similar. In the young child we rarely get these marked symptoms, and it is with these that academic discussion is exercised;—are they due to the same agent, the same micro-organism, as the adult cases? As a clinical fact "rheumatism *sine* joint lesions" leaves hearts just as crippled as the apparently severer forms, and it behoves the wary "medico" to grasp the dictum that the term "growing pains" is only rheumatism spelled in another way, and that a persistence of them will probably lead to future heart mischief.

Regarding the prevalence of acute rheumatism,

* An address given before the Clinical Section of the Lancaster Medical Book Club.

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contrary to popular opinion, the statistics and researches of Newsholme have clearly shown that it is not associated with a damp soil. This is strikingly seen by the statistics of countries where rheumatism is a notifiable disease; in Norway they had 67,907 cases in twenty-nine years. Records of Paris give the maximum number of cases in July; in Christiania it is stated to be in January, and Stockholm is the same; whereas in Philadelphia the minimum is September and the maximum in April. One factor in favour of an organismal origin is that it has been shown that rheumatic fever is especially prevalent when the subsoil water is low and at the same time the underground temperature high.

Heredity as a factor in the etiology is denied by Church, but in my own experience one often gets remarkable histories of rheumatic families, the difficulty being that the people do not always mean acute rheumatism. Only recently I got a very good rheumatic history from the friends of a patient in the institution, which extended back some generations, the chief affection being heart disease.

The effect of age is seen in the great prevalence amongst young adults, and although sex does not materially alter the figures, yet girls between ten and seventeen are very liable, and schoolmistresses ought therefore to be extra careful of their pupils at this period. No age is exempt; sucklings even have been known to have the complaint in its classical form.

As I premised, my remarks are very desultory, so I will now pass on to deal with the more practical side; the first question being, what is my own treatment of an adult case of acute rheumatism?

The patient has in all probability gone to bed, and so saved one the trouble of ordering him there. Clothing ought, of course, to be woollen, if silk cannot be afforded, and there should be plenty of garments to change with—a difficulty when treating the poor. For local application I find the so-called Fuller's lotion is as good as any; it is composed of subcarbonate of potash (Osler gives Sodæ Bicarb.) of the strength of ʒvj, with Tinct. Opii ʒj and glycerine ʒij, made up to ʒxij with water. But lately I have had great success with a new drug, official in the U.S. Pharmacopœia, and known as methyl salicylate, or artificial oil of winter-green. This is very soon absorbed, and may be detected in the urine in three quarters of an hour after being applied to a joint. The way generally recommended is to pour ʒj on a couple of folds of lint, and cover well with gutta-percha tissue, and then seal the edges with chloroform; but this latter is unnecessary if wool and a bandage be put over all. I may remark in passing that the yellowish protective won't do, as the drug dissolves the resin in it. My own practice is to wrap up one of the joints in this methyl dressing, and if several joints are painful, to use Fuller's lotion for the others, and change it about. I cannot bear out Yeo's statement in the new edition of his 'Manual,' that it should not be applied to acutely inflamed joints. Some persons object to the smell, but I find that when they feel the benefit of the drug they are only too glad to have it applied; the smarting is soon gone. The Bayer Company are now putting on the market a substitute for methyl salicylate which is almost odourless, and which is miscible with olive oil, or castor oil; I show you a sample of "Mesotan" that they sent me this week.

For internal medication we have a plethora of drugs. Of course there has been a serum prepared in Berlin, but it so far has not done any good to the best of my knowledge. In Nothnagel's 'Handbuch' Pribram has a list of seventy-five drugs in about sixty-seven pages that have been used for this complaint. The latest is called "Rheumatin," and in the 'B. M. Journal' "Epitome" for October 25th it is stated to be expensive, and to be lacking in unpleasant effects (one may therefore wonder if it is of any use), and in constitution is a combination of salicylic acid and salicylate of quinine.

Leaving aside quinine and opium, we are practically tied down to one or two methods of treatment—the old, or alkaline; or the new, or treatment by the salicyl compounds. Sir Samuel Wilks has caustically said that the "use of alkalies in this fever was to remove the idea of acidity from the mind of the doctor." Let me say a good word for the alkaline; its action, says Douglas Powell, is to increase the oxidation of the tissues, and promote elimination of morbid materials—surely a very useful action in such a disease as we are dealing with now. The new Osler is rather altered in this connection, and he seems to think that the cardiac complications are less with alkaline medication, but that the disease is not cut short nor the pain

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allayed. My belief is that one occasionally gets cases that absolutely refuse to improve until the patient is given Fuller's old prescription of potash in large doses frequently, until the urine is alkaline to test-paper. Recently one of our attendants was an excellent example of the value of this procedure.

When we come to consider the question of the salicylates, we are met by two factions—those who believe in the artificial salicylate, and those who prefer the natural products. The late Prof. Leech explained the superiority of the natural salt by the presence in it of a certain amount of methyl salicylate, and not to the impurity of the artificial salt. Methyl salicylate itself is used internally, but I have never done so on account of the nauseating taste; it needs to be emulsified or else given in capsules, and 150 ℥ have been so given in the twenty-four hours. It is excreted in the urine unchanged if in very large dose, but it generally behaves like salicylic acid and passes out as salicyluric acid. It is generally stated to be of no more use given internally than the pleasanter soda salt. Salophen and aspirin are other new salicyl derivatives that have been a good deal used; the latter is excellent at times, and has not the drawbacks of other salicyl drugs.

Osler is now much more in favour of the salicyl compounds; the supposed prevention of relapses is one point in their value. I use either the soda salt, or the ammonium salt made by double decomposition from the acid and Liq. Ammon. Fort., and for the acute stages I have not found salicin of equal certainty, but later on it is useful owing to its action as a bitter tonic on the stomach wall. Cardiac complications are not a reason for stopping the salicylates, but only for watchfulness. The full alkaline treatment requires the heart to be carefully examined. Beyond the usual ear disturbance I have only once come across a case of idiosyncrasy to soda salicylate, and that, too, after a small dose; the patient was very excited (as usual, a woman), and had hallucinations of sight and hearing, but these untoward symptoms soon passed off. Disturbance of the optic nerve, giving rise to troops of figures, has been reported several times.

Before passing on to say something about two complications of acute rheumatism—namely, endocarditis and chorea—I would say that I feel confident that appendicitis, tonsillitis, and rheumatism are allied, but one cannot always trace the connection to acute attacks, once more forcibly illustrating the dictum of Barlow, that we don't know really what constitutes the term rheumatism.

Taking first endocarditis, we are all alive to the fact that few cases, if at all severe, escape without some more or less serious heart involvement, and that we have to try and save the delicate heart wall from the baneful effects of the poison. Physiology helps us here as to our line of treatment: it tells us that the completely recumbent position diminishes the pulse-rate and relieves the pressure on the heart, and that it is only a question of arithmetic how much saving of strain is brought about by strictly keeping up this posture during the inflammatory period. Caton, whose name I shall have to mention again, keeps his patients in bed for eight weeks after the onset of endocarditis.

As to what constitutes the first stethoscopic evidence of the on-coming of the endocarditis, authorities are at variance; personally I hold strongly that the first sound at the apex becomes blurred, and that a systolic murmur follows in about forty-eight hours after. I have never come across any case where reduplication of the second apical sound was the forerunner of the murmur.

It is generally stated that endocarditis is less common where there is much joint effusion, but the probability is that the patient keeps so much the quieter that the heart is the more rested. As to the special treatment for the inflammation, it stands to reason that purely chemical stimulants, such as the various meat juices, are to be forbidden, and only milk and weak broths allowed in the dietary. Alcohol, I think, is hardly required; at any rate, it is contra-indicated at first. For local application repeated blisters the size of a florin, applied along the præcordial interspaces, following each by a poultice, as advocated by Caton, are very useful. He has had great success in *curing* his cases, but I suspect that the prolonged and perfect rest that he enjoins has a lot to do with the successful result.

Another thought occurs to me—why is endocarditis of the mitral valve much more common than the involvement of the aortic valves? We know that the left side of the heart is more usually involved, because venous blood has an inhibitory action on micro-organisms, and they must therefore journey further on. If there be bacilli in the joints

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they will be carried by the blood and deposited. Now normally there are no blood-vessels in the aortic cusps, but there are in the mitral, and this little histological fact seems to explain the marked preference for the mitral valve.

During recovery from the endocarditis either potassium or sodium iodide is useful, owing to their action in reducing blood-pressure.

Two further points are useful to note in treating rheumatic fever: one is the occasional involvement of the crico-arytænoid joints of the larynx, with symptoms of local pain and hoarseness, and necessitating abstinence from speaking on the part of the patient; the other point being that multiple neuritis may occur in a severe attack.

The importance of acute rheumatism in the young is forcibly illustrated by Sir Thomas Watson, who says, "I have known only three persons pass through acute rheumatism with an untouched heart prior to the age of puberty, and in two of them I am by no means certain that the articular disease was true rheumatism;" and with these statements Church agrees. There are very few people who will re-echo the opinion of Rosenstein when he asserts that the "disposal to endocardial affections, on the whole [is] smaller in childhood than after puberty."

We have to pass by many interesting features of rheumatic accompaniments, but I will close by some remarks on chorea. If any of you are wishing to read an excellent account of chorea, I can commend the little book by Sturges, or the great lecture of Trousseau's in the first volume of the 'Lectures on Clinical Medicine.'

Chorea is called by Dyce Duckworth and others "cerebral rheumatism," but we must keep in mind that this term is also applied to hyperpyrexia. It is far more common in females, but agrees with rheumatism in being rare before four years of age. I fear that we have made little progress either in the treatment or the pathology of the condition during the last twenty years.

Sydenham, about 1690, published the classical description of chorea, defining it as "a sort of convulsion which chiefly invades boys and girls from ten years of age to puberty, showing itself first by a certain lameness, or rather instability of one of the legs, which the patient drags after him like a fool." Sturges describes it very exactly as an exaggerated fidgetiness. Sydenham, of course, did not know its relationship to heart disease.

That it has a connection with rheumatism is known to all of us, but authorities are quite at variance as to what percentage follows on attacks of acute rheumatism. Comby holds that a rheumatic origin is uncommon, whereas Sée, in his prize essay in 1851, brought much evidence of the undoubted influence of rheumatism. Again, we are met by the difficulty of deciding exactly what complaints are included by different men when making up statistics of "rheumatism." Trousseau says that chorea rarely precedes rheumatic attacks, but follows in one third of the number of cases.

I need not trouble to discuss the various theories of the pathology of the affection, except in so far as to say that the apparently straightforward explanation of the occurrence of chorea from "emboli" derived from the mitral valve will not stand criticism. The great advocate of this was Kirkes, of Bart.'s, over fifty years ago, and in later years Hughlings Jackson espoused this theory. The latest idea is that the diplococci of Poynton and Paine block the brain capillaries much as malarial parasites do, and so give rise to chorea; undoubtedly these observers produced the symptoms of chorea in a rabbit, and post mortem they found the cocci in the brain capillaries in the motor cortex. Quite recently Fritz Meyer has also caused choreic symptoms, but could not obtain the organism in the brain. Goodhart does not think that there is any constant lesion, but that chorea is a functional disease, which, in our present state of knowledge of pathological processes, is equivalent to a confession of ignorance. One feature we always find in a choreic case—either bad nutrition or else a predisposition to nervous complaints; a fright is often the immediate cause of the attack.

Sydenham's treatment was by large doses of opium made up with Malaga wine and antispasmodics like cloves; he found what a lot of opium could be taken by persons when suffering from severe diseases. Little advance has been made since the time of Tuckwell, who wrote thus in 1876:—"An isolated ward, a good nurse, a large crib well padded round, plenty of good food without stimulants." I would enlarge on this by saying that chorea ought not to be treated in a

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children's ward; if you cannot get a separate room, put screens round the bed, and if violent, either use a slung hammock or else put netting over the top of the crib. Some people place the patients on mattresses on the floor. Mellin's food is a useful addition to the milk diet, as helping to supply some of the iron that the milk lacks.

Drugs are necessary if the case be severe, but it is difficult to believe that any one acts as a specific. Murray's arsenic treatment with heroic doses, *i. e.* even more than 15 ♏ thrice daily of Fowler's solution, taken in the middle of a meal, has hardly met with the success that he predicted. I believe he got the idea from a quack on his death-bed. On the Continent large doses of phenazonum have been fashionable; Comby gives 30 grs. in a dose thrice daily. Some prefer to use the acid solution of arsenic instead of Liquor Fowleri; this, too, is more common on the Continent.

For the restlessness I have found paraldehyde the best, and if the patient is not inclined to sleep the wet-pack often acts like a charm. Daily shampoo is very much enjoyed by the sufferer. I have not found ether-spraying of the spine of any good. If the case be a very severe one, there is nothing to replace chloral hydrate by mouth or rectum. Sleep is most important, as the movements then cease and the body gets an opportunity of repair and recuperation. I have only seen two fatal cases of chorea, and then nothing seemed of avail in stopping the exhaustion that carried them off. During convalescence iron and massage are both useful, but the desire to get the patient up too early must be combated. I show you a dietary of the Evelina Hospital for chorea cases treated by massage, and especially draw attention to the item of Kepler's malt given in lemonade, as being a very useful measure.

Gentlemen,—I cannot close these rather disjointed remarks without alluding to one other feature of chorea and rheumatism; I mean the "subcutaneous nodules," which are of such interest and so practically important.

The presence of nodules settles the rheumatic nature of any doubtful case, and they are also of prognostic import, for they imply the presence or the immediate onset of heart mischief. The more numerous they are the worse the outlook, especially if chorea has supervened. Relapses often bring out a fresh crop, and that they are connected with infection is borne out by the researches of Poynton, who found the diplococci in them. They are not strictly "fibrous nodules," under which name they usually go, but much more fibrinous in structure, and often evanescent and not easy to dissect out.

My own inquiries seem to show that the prevalence of these nodules varies with different districts; I have not succeeded in finding any on any patient in the Royal Albert, yet many of these are very rheumatic subjects. Rarely one finds them in the adult. Feeling is better than seeing when searching for them, and the olecranon and malleoli, the front of the tibia, and the cervical spines and occipital crests, are the seats of election. They are painless, and may be multiple or single, and, if on the tendon sheaths of the front of the forearm, are very freely movable.

In this rapid review of rheumatism and its concomitants I have just skimmed over a wide field; the gaps, perhaps, other members with more experience than I possess can fill up.

In closing, let me quote the words of a layman, and also those of a medical man:—"Life," said Napoleon, "is a fortress. . . . . Why throw obstacles in the way of its defence? Its own means are superior to all the apparatus of your laboratories." Our scientist says, "The microbe is nothing, while the organism in which it thrives is the main factor."

Mr. President,—Does our experience of rheumatism bear out these ideas? The medical science of to-day will, I think, hardly assent to the last aphorism I have quoted.

A VERY useful addition has been made by Messrs. Burroughs Wellcome & Co. to their list of "Tabloid" hypodermic products, *i. e.* strychnine sulphate of the strength of $\frac{1}{60}$th of a grain in each tabloid. The five hypodermic products of strychnine sulphate now on the list range from $\frac{1}{30}$th of a grain to $\frac{1}{80}$th. These hypodermic tabloids provide a ready and reliable means of administering strychnine in cases of emergency, such as those in which heart failure has to be reckoned with. The well-known fact that patients almost dead from failure of the heart in the course of chronic cardiac disease may sometimes be restored by the subcutaneous injection of strychnine makes these tabloids of great value, not only by reason of the accuracy of dosage that is assured, but also because of their free solubility and portability.

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CONGENITAL CEREBELLAR ATAXIA.

(CEREBELLAR DIPLEGIA.)

By F. E. BATTEN, M.A., M.D., F.R.C.P.,

Assistant Physician to the Hospital for Sick Children, Great Ormond Street, and to the Hospital for Paralysed and Epileptic, Queen Square.

I PROPOSE to bring under your notice to-day a series of cases showing marked ataxia which there is reason to believe is of cerebellar origin. The following definition of the affection may be given:—It is a disease of congenital origin starting in earliest life and characterised by marked unsteadiness of the head, trunk, and limbs, alteration in articulation, unsteadiness in sitting, in standing, and in walking, and by difficulty and slowness in swallowing. The general tendency of the disease is towards recovery, so that a child who through ataxia is unable to walk at two years old will by the age of four have learnt to walk, although probably still somewhat unsteadily. The disease must be carefully distinguished from *hereditary cerebellar ataxia* (Marie and Sanger Brown), and from *hereditary ataxia* (Friedreich's disease). The diagnosis from these two diseases will be discussed presently.

The following series of eight cases, which I believe to be examples of this disease, have come under my notice during the past few years:

D. S—, æt. 4, was first seen in February, 1899. She was the first and only child, and there had been no miscarriages. The labour had been prolonged, and instruments were used to deliver the child. The mother was in good health during pregnancy. The child was brought to the hospital because she could not walk. She had learnt to use her hand when twelve months old, and she could talk, but the articulation was defective, and she was difficult to understand. She had always had difficulty in swallowing, and had to be fed

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very slowly; the difficulty was greater with solids than with fluids. She was intelligent and clean in her habits, and had never had any illness.

On examination the child was well formed and nourished; she could stand and support her weight on the legs, but was unable to walk, and always tended to fall backwards. When an attempt to walk was made the legs were thrown about in a wildly ataxic manner. The head and trunk were in constant movement, and on attempting to grasp an object with the hands the most marked inco-ordination and intention tremors were exhibited. The head was well formed, measuring 19½ inches in circumference, and there was no deformity of the skull. The movements of the eyes were good in all directions, and there was no nystagmus. The optic discs were normal. The hearing was good; the tongue protruded normally, and the hard and soft palate were normal. The back was straight, and there was no bony deformity either of the limbs, feet, or hands. The power of movement of the limbs was good, and there was no rigidity. The knee-jerks were brisk and equal. There was no ankle-clonus, and the plantar reflexes gave fairly regularly extension of the great toe. In October, 1899, she was seen again, and on this occasion could walk with great difficulty and showed the same tendency to fall backwards. In February, 1890, her walking had improved, but she still tended to fall backwards. The head measured twenty inches in circumference. The knee-jerks were brisk, and the plantar reflexes were noted as not giving a definite extensor response, *i. e.* the big toe extended, the small toes flexed. In May, 1900, her walking had greatly improved; she could walk by herself, but still tended to fall backwards. There was much less inco-ordination of the hands, but the articulation was still very defective. In March, 1901, she walked fairly well, with the trunk held forward, rather like a case of paralysis agitans. The inco-ordination of the limbs and trunk was still present, though considerably less than when first seen. The knee-jerks remained active; the plantars gave rather varying responses, sometimes extensor, sometimes flexor.

The child when last seen was six years old. I lost sight of her, as she and her parents went to America.

N. N—, æt. 4¾, was first seen in July, 1901, and was under the care of my colleague Dr. Still, to whom I am indebted for permission to refer to the case. She had always had difficulty in walking, and only began to get on her legs when eighteen months old. She had difficulty in swallowing and had to eat very slowly. She was the eldest of three children; the second was healthy and the third had died of diarrhœa and vomiting. The labour was difficult and instruments were used, and the mother suffered from eclampsia during labour. As a baby and up till two years ago the child used to keep the head to one side, so that the left ear was approximated to the left shoulder. She was a bright, intelligent child. She walked in an ataxic manner with the legs far apart, and talked in the slow, monotonous manner which is so characteristic of these cases. There was no squint, no nystagmus, but the head was shaky. All the facial movements were good, and the tongue protruded straight. The grasp was good, but there was an intention tremor of both hands. The trunk exhibited the same shaky movements. There was no lateral curvature. The gait was ataxic. There was no rigidity of the legs, and all movements were well performed. The knee-jerks were active; there was no ankle-clonus, and the plantars gave a doubtful response, which was usually flexor in type.

H. J—, æt. 3 years and 3 months, boy, was first seen in June, 1902, and was brought to the hospital because he had not learnt to walk.

He learnt to talk at two years of age, and was bright and intelligent. He was unsteady with his hands, and could not stand unless his back was against the wall. He took food well and swallowed naturally. He was clean in his habits, but sometimes had an accident, which greatly annoyed him. He was not passionate. He was the first of two children, and there had been no miscarriages. The mother stated that she was not in good health during pregnancy, but the labour was natural and rapid. The child was a well-nourished, intelligent little boy, with the slow, monotonous articulation characteristic of this affection, but he had in addition a lisp, so that he omitted the first consonant of a word; for example, he said "oor" for door. He stood unsteadily and seemed always likely to fall. He walked with the knees flexed and close together, like a cerebral diplegia of a mild type, but at the same time there was marked

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inco-ordination. The legs could not be said to be rigid, although the mother stated that they were so at times. The knee-jerks were present, not very active; there was no ankle-clonus, and the plantars gave a flexor response, though it was a response which was neither easy to obtain nor definite in character. All the movements of the face were good; the movements of the eyes were good; the pupils were equal and reacted well to light; there was no nystagmus. The child sat up fairly well, but there was almost constant irregular movements of the trunk. There was no curvature of the spine. There was no rigidity of the arms; the grasp was good, and there was rather marked inco-ordination of both hands on attempting to pick up a penny, but the intention tremor was slight.

The child was seen again in December, 1902. He had learnt to walk, but the gait was still ataxic. He could sit up, but could not balance himself in a chair without sides. The mother noticed that when putting on his boots the right great toe often got "hitched back," so that she had to disengage it before the foot would pass into the boot. The knee-jerks were brisk; there was no ankle-clonus, and the plantar reflexes were flexor in type. The child was seen again in March, 1903. He had made further improvement, though still somewhat ataxic. The knee-jerks were active. The plantar reflexes were flexor in type.

R. C—, æt. 6, was first seen in July, 1900. She was the sixth of seven children, all the others being healthy. The labour was prolonged and was followed by flooding. The child began to walk when four years old, but tumbled about a good deal. She learnt to talk when two years old, and had always been sharp and intelligent. She was clean in her habits. She was very slow in eating and had difficulty in swallowing.

When first seen she walked in a very inco-ordinate manner, and was very liable to fall backwards. There was more inco-ordination of the right arm and leg than of the left. There was no rigidity of the limbs. The speech was slow and drawling, but the individual words were easily understood. There was no nystagmus, and the optic discs were normal. The knee-jerks were active; there was no ankle-clonus, and the plantar reflex gave flexor responses.

In March, 1901, the child had made very considerable improvement; she could walk fairly well, but was still unsteady. The knee-jerks were brisk; there was no ankle-clonus, and the plantars gave an indefinite response. In July, 1902, she was again seen, and had made further improvement.

In March, 1903, she was nine years old, but her mother stated that she was more like a child of five. She had been to school for three to four years, but had not made much progress. The inco-ordination of the hands and legs was very much less, and this the mother attributed to the use of a special kind of board. The articulation still maintained its slow, drawling character. The knee-jerks were increased.

Florence J—, æt. 2 years and 8 months, was first seen at the Children's Hospital, Great Ormond Street, in July, 1900, when her mother brought her because she was unable to walk and unable to talk. She was the elder of two children. The mother had been in good health during pregnancy and the labour was natural. She had had one miscarriage since the birth of the child. The child had had fits while teething. The child was well nourished, had a well-formed head, but was mentally defective. There was an internal strabismus which was said to have existed since birth. There was no nystagmus. The legs were somewhat rigid when attempting to walk, but were not rigid at other times, and they were thrown about in the most inco-ordinate manner, the child being quite unable to stand alone.

The same inco-ordination exhibited itself with regard to the movements of the hands, the right side being more affected than the left. The knee-jerks were not easy to obtain; there was no ankle-clonus, and the plantar reflex gave a flexor response. The child was not clean in her habits; she was passionate, but not destructive. She had not learnt to talk.

In November, 1900, the walking had improved, but the gait was still very inco-ordinate and high-stepping. During this month she had one attack of loss of consciousness; the eyes were fixed; the limbs became perfectly flaccid, and there were no tonic or clonic movements. The loss of consciousness lasted twenty minutes, and at the end of the attack she vomited. The knee-jerks were just obtained, and the plantars gave a flexor response.

In September, 1901, the general condition of the child had improved. She was then three years

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and nine months old. She still walked with a very wide base, and had worn out the tips of her boots. There was much less inco-ordination of the hands. The legs were flaccid. The knee-jerks were just obtained; there was no ankle-clonus, and the plantar reflex gave extensor responses. She could not say any words, but she was much better in her general behaviour, and could make her wants understood.

In March, 1902, further improvement had taken place. The ataxia was still present, but was less marked, and the knee-jerks were just obtainable.

In March, 1903, when she was five years old, further improvement had taken place. She could only say a few words, such as "mum," "dada," and "Lily." She walked in a most ataxic way, with the legs far apart, but although so ataxic she seldom fell. The inco-ordination of the legs was much greater than that of the arms. The legs were not rigid; the knee-jerks were active; there was no ankle-clonus, and the plantar reflex generally gave an extensor response.

F. W—, æt. 5, was first seen in July, 1900. He was the third of three children, of whom the first two were quite healthy. The labour was natural, but the mother stated that she was not in good health during pregnancy.

The child learnt to talk at a year old. He had never learnt to walk. He had had no fits and no serious illness. He was a poorly developed child of weak intelligence. When attempting to stand or walk he was very unsteady, and walked with the legs wide apart. He was unable to stand with the feet together. There was a marked internal strabismus, due to loss of power in both external recti. The pupils were equal and reacted to light. Nystagmus, which had been present since birth, was almost constant, and he had squinted since a few months after birth. No satisfactory examination of the discs could be obtained. He saw fairly well. The back was straight. The legs were thin and not rigid; there was no pes cavus. The knee-jerks were present; there was no ankle-clonus, and the plantars gave an indefinite extensor response. There was slight inco-ordination of the arms.

In July, 1901, the boy's condition had very much improved, but there was still considerable ataxia of the legs.

In March, 1903, still further improvement had taken place; he could walk fairly well, but was still unsteady. The condition of the eyes remained unaltered. The articulation was still very defective, and he was difficult to understand. The inco-ordination of the hands and legs was very much less. The knee-jerks were active, and the plantars both gave extensor responses.

J. B—, a girl æt. 3, was first seen in October, 1900, at the Children's Hospital, Great Ormond Street. She was brought because she was unable to walk, was unable to talk, had screaming attacks, and was not clean in her habits. She was the only child; there had been no miscarriages, and the mother was in good health during pregnancy. No instruments were used, and the labour had lasted four hours. The child had never had any fits. She was well developed and well nourished. She was unable to walk, but could support the weight of the body on the legs, and if held up could make some movements forward. She tended to fall back. There was marked inco-ordination of the legs and arms. There was no rigidity. The knee-jerks were present, equal but not active; there was no ankle-clonus, and the plantars gave flexor responses. The movements of the eyes were normal; there was no nystagmus. The discs were normal, but there was a stippled appearance of the macula. The head was rather small, measuring eighteen inches in circumference, and was flattened in the occipital region. The back showed some slight lateral and dorsal curve.

In March, 1901, some improvement had taken place. She walked better, but the gait was still very inco-ordinate. The knee-jerks were brisk; there was no ankle-clonus, and the plantar reflexes gave flexor responses. She was still dirty in her habits. She had learnt to say a few words.

March, 1903.—The child still remained in a very backward state, and no improvement had taken place in her walking, which was still wildly ataxic. She had had some epileptic fits. The knee-jerks were still brisk, and the plantar reflexes gave flexor responses.

L. J— was first seen in June, 1894, æt. 1 year 5 months, when she was first admitted into the Children's Hospital, Great Ormond Street, under the care of Dr. Lees, and to him I am indebted for permission to refer to the case.

The child was a fine, healthy baby at birth, but when six weeks old it was noticed that she was constantly nodding her head, and that the eyes

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moved from side to side. As soon as the child was old enough to take an interest in its surroundings her mother noticed that her arms, especially the left, which she used most, would make wide excursions before seizing the object she wished. It was also noticed that she had great difficulty in putting her fingers to her mouth. The child could not stand on account of the jerky movements. She was the youngest of a family of eight—four boys and four girls. All the other children were healthy, and the mother and father were healthy. The labour lasted five hours, and instruments were used. The child was well nourished and appeared healthy. While lying in bed she was fairly steady, constant nystagmus being the most noticeable feature. There were occasional sudden jerky movements of the arms. When the child was placed in sitting position the whole body shook, the head nodded, and the hands were moved about in an ataxic manner, and marked intention tremor was present on attempted movement. The head was well formed and the child took notice of things. The pupils were medium size and reacted well to light. There was constant lateral and rotatory nystagmus. The optic discs were normal. When attempting to walk with aid she showed a very good idea of putting one leg before the other, but was quite unable to stand alone. Arm-jerks were present, but were not marked. Knee-jerks were active. Ankle-clonus was present, rather more marked on the left side than the right, and the plantars were active (the note was taken in the pre-Babinski days).

The child was seen as an out-patient again in 1898; all the symptoms were as marked as on the first occasion when seen. She had learnt to talk, and spoke in a slow, monotonous manner. The legs were now rigid, and she was quite unable to walk.

The child was again admitted to the hospital in 1901, when eight and a half years old. She had grown and was well nourished. Her mental condition was good, considering her disabilities; she would answer simple questions and obey directions. Her articulation was slow and monotonous, with divided syllables. Her vision seemed fair. The nodding movement of the head, the nystagmus, the intention tremor of the arms, and the unsteadiness of the limbs and legs were all present and as marked as on the former occasions when the child was seen. The legs, however, were more rigid, strongly adducted. The knee-jerks were exaggerated; ankle-clonus was present and more marked on the left side than the right. The plantar reflex gave extensor responses, but not to a marked degree. There was no pes cavus or other deformity of the bones.

These cases, although they differ from one another in many particulars, all show, or have shown, a very marked degree of ataxia, such as is commonly associated with lesion of the cerebellum; and the marked ataxia in association with absence of rigidity are the two chief reasons for regarding the cases as probably cerebellar in origin.

The cases have with design and intention been arranged in the order in which they are related, so as to bring into prominence the fact that while in the first case there is reason to believe that the pathological condition is almost limited to the cerebellum, in the succeeding cases other symptoms present themselves which most certainly point to some implication of the cerebral hemisphere, and in the last case the rigidity is such that the preponderating pathological process lies rather in the cerebrum than in the cerebellum.

Freud has described two cases in which the symptoms closely resemble those described in the present series of cases, and in assigning these cases to the group "cerebral diplegia" he points out that none of the symptoms seen are foreign to those found in cases of cerebral diplegia. Nystagmus not infrequently occurs; strabismus is very frequent, as also are unsteadiness and tremor of the hands and the monotonous slow speech. Some cases of cerebral diplegia show no mental defect.

These symptoms have, indeed, been attributed to a defect in the cerebrum, but some of them are such as are known to be associated with lesions of the cerebellum, and it seems probable that they are rather cerebellar manifestations than cerebral.

From the clinical side the evidence with regard to the symptoms produced by atrophy of the cerebellum is most discordant; in some cases there have been no symptoms, whereas in others symptoms have been marked. One of the most typical cases of a progressive cerebellar ataxia is that reported by Dr. Donald Fraser, who describes two cases in a brother and a sister between twenty and thirty, which gave rise to a group of symptoms strongly suggesting cerebellar disease, and in which at the autopsy in the male patient atrophy of the

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cerebellum was found. The disease in these two cases started about the third year of life, up to which time the children appeared to be of normal development, and by the sixth year the symptoms had become marked. The weight of the cerebellum was about half the normal weight, and the atrophy affected the grey rather than the white matter.

The experimental evidence of the symptoms produced by ablation of the cerebellum is derived from the work of Luciani. The symptoms are: general unsteadiness, strabismus, spasms of the muscles of the neck, flexions of the fore-limbs, weakness, hypotonia, and unsteadiness of the posterior extremities, all of which symptoms tended to pass off, so that after a few weeks there was but little to distinguish the animal from one with an intact cerebellum. Luciani, however, has shown that if the cerebral cortex be damaged in the region of the gyrus sigmoidea, then the symptoms seen after removal of the cerebellum persist.

Have we not in this experiment the explanation of a clinical fact that is well known, that there may be atrophy and even complete absence of the cerebellum without there being any symptom pointing to cerebellar disease? So in the present group of cases, had the cerebrum been perfectly normal, no symptom of the cerebellar disease would have been manifest, but the defect of the cerebrum brings into prominence the cerebellar symptoms.

In those cases of the present series in which recovery has been most perfect, but little evidence is found of any affection of the cerebrum; whereas in other cases mental impairment to a greater or less degree is present, the plantar reflexes gave extensor responses indicating possibly some affection of the pyramidal tract, and in one case actual rigidity of the legs was present.

That these cases belong to the class of "diplegia," using the word in its widest sense, is probable, but they should be classed not as cerebral diplegia, but as cerebellar diplegia, fully realising that between what may be regarded as a typical case of *cerebral* diplegia with the rigid legs, active knee-jerks, and extensor responses of the plantar reflexes, and a cerebellar diplegia with marked ataxia and flaccid limbs, there are a series of transitional cases which partake of the symptoms of both groups.

Further evidence may be derived from animals, not from experimental sources, but from observation of actual disease. The following report on two kittens is so admirably written by Dr. Herringham and Dr. Andrewes that I will repeat it in their own words:

In May, 1887, a half-bred Persian cat belonging to the Sister of Hope Ward in St. Bartholomew's Hospital produced a litter of four kittens. These appeared healthy at birth and grew well for a time. When they began to walk, they at first moved like healthy kittens, but soon one of them was noticed to be weak on the legs. Another was soon afterwards similarly affected, and these two gradually became so incapable of movement that they were destroyed while only a few weeks old. At the time of their death the remaining two appeared quite healthy; but not long afterwards, first one and then the other showed symptoms of the same weakness, which progressed until in September they came under our notice. It is therefore certain that the symptoms did not date from birth, that they did not come on at the same time in all the kittens, and lastly, they were of a progressive character.

When we saw the kittens in September one was much more incapable than the other, but the morbid symptoms were so exactly alike that one description embraces both. The nutrition of the body was good, the coat sleek and clean. The head and eyes were moved well and naturally, and there seemed no loss of vision. One of them examined with the ophthalmoscope showed a natural fundus. The ears, whiskers, cheeks, nose, tongue, and tail were moved naturally. The cats lapped milk and mewed as usual. Micturition and defæcation were performed properly. There was, however, great loss of power to maintain equilibrium, both at rest and in motion. When sitting down the beast held the fore-legs wide apart so as to increase its supporting base, and even then a slight movement would suffice to throw it over. In walking it seemed as if either the fore-part of the cat or its hind part could have walked separately, but as if they were badly joined together; the whole hind-quarters swayed and tottered from side to side and often fell over, thus dragging the fore-part with them, making locomotion very difficult and uncertain. The little delicate movements of the fore-paw, which are peculiarly feline, were well performed, and the rapid motion of scratching the head with the hind foot was natural.

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The first cat was killed when four months old, and the second when eight months old, and when the skull was opened the eye was at once struck by the smallness of the cerebellum, and especially its middle lobe. The grey matter was more affected than the white, and the most marked changes were in the granular and molecular layers. The cells of Purkinjë, although irregular in arrangement, appeared normal in size and in number.

A second observation on deficiency of the cerebellum in animals is that of Dr. Risien Russell in a puppy.

The animal was one of a litter of puppies all similarly affected, though born of healthy parents, both of whom were prize dogs. During life the puppy had manifested such marked inco-ordination that on attempting to walk the animal fell, sometimes in one direction, sometimes in another, there being no one direction in which it was inclined to fall more frequently than in any other. When standing, or even when sitting, there was almost constant oscillation of the head and trunk. The puppy was killed and the cerebellum was found reduced to about three fourths the size of the normal. Microscopically changes were found in the molecular and granular layers, and also to a still more striking extent in the cells of Purkinjë. The appearances met with in the cerebellum in this animal made it clear that the defects were of congenital origin. It is interesting to observe so much disturbance of equilibrium, for an animal deprived of even the half of the cerebellum, experimentally, regains its powers of equilibrium in a remarkably short time.

Other cases in animals of a similar nature might be quoted, but the above most carefully recorded instances are sufficient to show for the present how similar the symptoms were in these animals to those in the present series of cases.

I unfortunately cannot describe to you the exact pathology of the condition which is present in the series of cases, for an opportunity has never arisen for a pathological investigation.

Diagnosis.—The diseases to which the present series of cases bear the most strong resemblance are hereditary ataxia (Friedreich's disease) and hereditary cerebellar ataxia (Marie and Sanger Brown), and although there are some differences in the symptoms, yet it is in the course of the disease that the most striking differences occur. In hereditary ataxia (Friedreich's disease) the leading feature may be said to be slow and progressive ataxia of the four limbs, usually attacking several children of the same family, beginning about the tenth to fifteenth year of life, affecting first the legs and gradually extending to the trunk and arms, with irregular movements of the trunk and head, difficulty in articulation, nystagmus, deformity of the spine and feet, and absent knee-jerks.

It may therefore be said that the progressive course of the disease, the age of onset, the deformity of the spine and feet, and the absent knee-jerks constitute the most striking features in the differential diagnosis between hereditary ataxia and congenital cerebellar ataxia.

In hereditary cerebellar ataxia (Marie and Sanger Brown) the leading features of the disease may be said to be its markedly hereditary character, traceable through several generations; its onset between the ages of fifteen and thirty-five; the inco-ordination of the limbs, first to be noticed in the legs, but afterwards extending to the arms, face, and organs of speech; atrophy of the optic disc; alteration in articulation; exaggerations of the knee-jerks; and ankle-clonus. Nystagmus and deformity of the feet are usually absent.

It may therefore be said that the course of the disease, the age of the onset, and the optic atrophy are the most striking features in the differential diagnosis of *hereditary* cerebellar ataxia and *congenital* cerebellar ataxia.

Disseminated sclerosis in its symptoms clinically most closely resembles congenital cerebellar ataxia, and there is but little doubt that cases of this disease have been regarded as cases of disseminated sclerosis. Disseminated sclerosis is a most rare disease in children, and the fact that the symptoms in congenital cerebellar ataxia are stationary or retrogressive rather than progressive would be the most distinguishing feature between the two diseases.

Diphtheritic paralysis, acute vascular lesions of the cerebellum, cerebellar tumour and abscess, may all give rise to ataxia of a cerebellar type, but the history of recent onset and the acute symptoms would at once make the diagnosis easy.

Cerebral diplegia with choreiform movements bears possibly some superficial resemblance to this disease, but the movements are choreiform rather than ataxic, and there is not the marked

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intention tremor and ataxia that are seen in this disease.

Prognosis.—The importance of a correct diagnosis and the appreciation of the nature of the cases here described is considerable, for the tendency of the disease is towards recovery, whereas in the other form of hereditary ataxia the tendency of the disease is progressive. It may be stated that children in whom this ataxia is present and marked when two years old, and in whom there is no marked mental defect, will learn to walk and will walk fairly by four or five. They will learn to talk, but probably always in a slow, monotonous way.

Treatment is of considerable importance.

First, with regard to co-ordination of the fingers and hands, a solitaire board or one of the numerous forms of mosaic puzzles forms an excellent training.

Second, with regard to the legs, a modified Fraenkel's exercise board is of great service, and frequent exercise in walking. Passive and resistant movements also do great good.

I hope I have demonstrated to you this afternoon a class of cases which have hardly obtained the recognition which they should, and which may be called congenital cerebellar ataxia, or cerebellar diplegia. I hope that at some future date I may be able to demonstrate to you the pathology of a disease of which I can at present but give you the clinical features.

Failure of X Ray in Hodgkin's Disease.—The brilliant results obtained from use of X radiance in superficial cancer are not duplicated in Hodgkin's disease. In a recent communication Dr. Francis H. Williams reports concerning his most hopeful case (out of three in which he has used the rays) that, although surprisingly rapid improvement was observed for a time, at the end of six months the glands had enlarged again, and the patient's general condition was no better than it was at first, in spite of the fact that the treatment had been continued.—*American Practitioner and News*, February, 1903.

THE PHILOSOPHY OF POSTURE AND AMBIDEXTERITY.

By E. NOBLE SMITH, F.R.C.S.Edin.,
Senior Surgeon to the City Orthopædic Hospital, London.

In the streets of every city and large town, and even in many country districts, we see cripples who show the results of a preponderating one-sided action. This one-sided action may be brought about by the crippling effect of paralysis of a foot or leg or side of the trunk; inequality produced by shortness of one limb from joint or other disease, or from original defect; inequality in weight, as when an individual has lost an arm, etc.

The conditions named and others of a like kind are bad enough in themselves if suddenly imposed upon an individual, but worse results follow if the deformity produced is not counteracted by surgical treatment. When the body is thrown out of its proper equilibrium and remains so, increasing deformity must ensue. Slowly but surely the various joints of the body give way, and even the bones adapt themselves to their changed position. A person who has lost some power or is lame in the right leg has, when standing upright or walking, to lean his body over to the left side (Fig. 1), so that the centre of gravity shall fall chiefly upon the left leg, instead of directly between the limbs.

Fig. 1 (seen from the back).

In thus walking the equilibrium of the body is maintained by balancing between the head and shoulders thrown to the left, and the lame leg thrown outwards to the right (supposing the lame leg to be the right). This position is facilitated by the use of a stick on the sound side. Anyone

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whose right leg is shorter than the left must do one of two things—either he must raise the short leg by standing on the toes of that side, or, which is more usual, he must lower the right side until his right foot rests evenly on the ground. This produces obliquity of the pelvis, and tilts the spine to the right in the direction of the dotted line A B. No one, however, could walk about in this position, and therefore the spine is curved to produce equilibrium of the upper part of the body. It necessitates his curving the lower part of his back to the right (C) and *compensating* such bending by curving the upper part of his back to the left, and perhaps the neck also to the right,—that is to say, the equilibrium is attained by diffusing the tendency to deflection of the body into several curves. These curves, being constant whenever the patient is standing or walking, eventually become per-

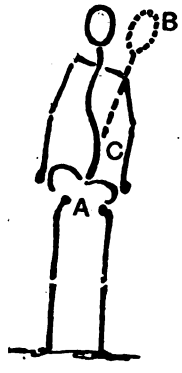

FIG. 2 (seen from the back).

manent from the joints of the spine and even the bones themselves changing their shape.

There are some cases of short leg which have a contrary influence. When the hip-joint is very weak or painful there may be an inclination to lift the leg and with it the hip-joint on that side rather than depress it. A good example of this is seen in that peculiar condition known as congenital displacement of the hips. Children are sometimes born with one or both hips displaced, the sockets and the heads of the thigh-bones often being imperfectly developed. When the child begins to walk it is found he is lame, or at least walks awkwardly—limps or "waddles."

If one leg alone is thus affected the limb will be apparently short, but the child does not depress that side of the body. Upon the contrary, he will make an effort to raise that limb and throw it forward in walking because there is no stability in the joint, and the afflicted child prefers to raise that hip while bringing the affected leg forwards.

These few instances given could be greatly added

FIG. 3 (seen from back).

to, but they will suffice to illustrate the fact that persistent irregularity of posture must inevitably produce deformity; wry-neck, irregular shoulders, curvatures of the spine, irregular hips, irregular gait, knocked knees, crooked ankles, flat feet, are examples of the most frequent results of a one-sided posture and action of the body; whereas, upon the other hand, if these affections occur from some independent condition they will act as the cause of such one-sided position.

In wry-neck, say to the left, the head is held

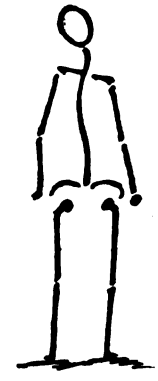

FIG. 4.

down (probably by a contracted muscle) towards the left shoulder. This position causes the neck to curve towards the right (see Fig. 4). Now it would be very inconvenient or impossible for the patient to keep the remainder of the spine per-

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fectly straight while the neck remained curved, and, therefore, unconsciously the lower part of the spine is curved in the opposite direction to *compensate* for the strong neck curve. In these cases the shoulder on the contracted side is often drawn up

FIG. 5 (seen from back).

towards the head, further complicating the irregularity. Irregular shoulders are almost always produced by curvature of the spine, but it is possible to have contracture of muscles or other conditions, causing the patient to hold the shoulders unevenly; under the latter circumstances the right arm, for instance, being held high, causes a spinal dorsal

FIG. 6. FIG. 7.

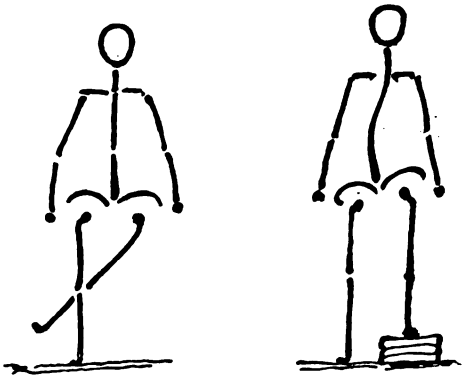

FIG. 6.—Forced adduction from contracted adductor muscles, etc., necessitates tilting of the pelvis in order to bring the contracted limb into a vertical position, and then the affected leg is raised from the ground (Fig. 7), and this necessitates curving of the spine.

curve to the right (Fig. 5). This will be dealt with further presently.

Irregularity of the hips is generally produced by a short leg or curvature of the spine or both combined, or from forced adduction of a limb (Fig. 6), or forced abduction. Irregular gait has, of course, many causes, but the results are similar,—that is to say, a constant or almost constant uneven action of the body inevitably leads to crookedness being produced in the body.

Now just as morbid conditions such as those above referred to must produce these one-sided effects, so may acquired bad postures have a like influence.

For ages medical men have complained about the postures peculiar to work at school, and we have deplored the production of spinal curvature and other deformities as the result of such postures. Drilling and gymnastics have done something to counteract these influences, but have by no means prevented the evil; special seats and desks have also been beneficial, but are not infallible; the improvement of dietaries and attention to the general hygiene of the pupils have rendered them less liable to such bad effects, but yet the deformities continue to occur.

As far as postural deformity is concerned there is one prime factor which underlies all others.

That factor is right-handedness (unidexterity). The bad habit of neglecting to develop the faculties of the left hand, and therefore the preponderating use of the right hand in writing and drawing, and in all school work, and in nearly all games, has the effect of causing the upper dorsal region of the spine to curve towards the right, and in course of time such persistently repeated curving causes radical changes in the shape of the joints and bones of the spine.

Although there are many causes of spinal curvature, predisposing and exciting, the one-sided influence of right-handedness is so great that it acts most powerfully in promoting and increasing lateral curvatures of the spine.

I have elsewhere and on many occasions written and lectured upon the treatment of spinal curvatures, and the consideration of such treatment does not belong to the subject of this paper, but I must refer incidentally to one point.

In the special muscular exercises which I have devised for acting directly upon the curves, I have brought into action the muscles which unite the *right* arm to the spine, to correct a *right* dorsal curve. Now it might be asked, "How can exercise of the right arm correct a *right* dorsal curve if a

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preponderating use of that arm has caused or increased that curvature?"

The explanation of this apparent inconsistency is that the excessive use of the right arm influences the spine detrimentally by *the postures which it induces.*

In using the right arm in excess of the left, all the muscles which unite the arm to the body are used indiscriminately.

In my plan of treatment the muscles which unite the arm posteriorly and the shoulder-blade to the spine are especially and almost exclusively brought into action, whilst the patient rests recumbent in a certain position. The recumbent posture tends to correct the curve, and the exercise limited to the few selected muscles tends to rotate the spine into a correct, or at least an improved position. For a fuller description I must refer the reader to other of my writings.* I merely discuss the subject here, briefly, because the above question might arise.

In lateral curvature of the spine a dorsal curve to the *right* exceeds greatly, in proportion of numbers, curvature in the opposite direction. In 200 cases which I examined and analysed in this particular a dorsal curve to the right was present more than twice as frequently as one to the left. In another 118 cases which I have analysed I have found the proportion of right dorsal curves as 5 to every left curve; but I should add that in a large majority of the left curves there has been a distinct special cause for the direction, such as a short leg, a wry-neck, congenital deformity, etc., and when there has been no such special cause the direction has been almost invariably to the right. In an analysis of 1000 cases made at an orthopædic hospital the proportion was 5 on the right to 1 on the left; while Bouvier, a French surgeon, found the proportion to be 7 to 1. The most probable preponderating cause of the dorsal curve being chiefly to the right is the prevalence of right-handedness. Upon the other hand, there are so many other definite causes of curvature that we must not expect to find all patients suffering from left curvatures to be left-handed. For instance, anyone who very frequently carries a heavy weight with the right hand (Fig. 8) must gain his equilibrium by leaning over to the left side, thus producing a dorsal curve to the left and a sharp lumbar curve to the right.

A nursemaid who carries a child always on the left arm produces dorsal curve to the right and lumbar to left.

I have seen a girl of twenty, with a strong left-handed curve, whose position at her work of hat-making at Luton necessitated her bending into this position although she was right-handed. In a case of congenital defect of the right forearm, where the patient was necessarily left-handed, a left curve existed, but this seemed to be chiefly the effect of holding things with the right stump pressed hard against the body as well as the left-handedness, and there were the two influences acting in the same direction. Wry-neck, as already stated, causing the head to be held down to the left side, will cause a curve to the right in the neck,

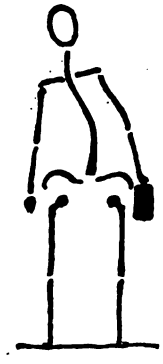

FIG. 8 (seen from behind).

and a dorsal curve to the left. Shortness of one leg, when the hip-joint is not in itself weak, will produce a lumbar curve towards the short side and a compensating dorsal curve in the opposite direction.

The outcome of these inquiries shows that it is the *preponderating* influence that determines the direction of a curve, but, if other things are equal, right-handedness remains a very strong incentive to a right dorsal curve. A great deal of women's work, such as needlework, flower-making, etc., causes a dorsal curve to the right. It should be understood, however, that with curvature of the spine there is usually a predisposing cause of general weakness or weakness of the joints of the spine, but that curvature may also be produced when no weakness exists, *if the exciting cause be very strong.*

* 'Curvature of Spine,' 4th edit., Smith, Elder & Co., London; "Exercises in the Treatment of Lateral Curvature of the Spine," 'Lancet,' July 7th, 1900.

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The influence of right-handedness is far reaching. For instance, there is the deforming influence already described, and there is especially the limitation of the usefulness of the individual by the neglect of the left hand. This is especially seen in most trades and in professions where manual capacity is necessary. In surgery the need of equal ability with both hands is so obvious that many surgeons become ambidextrous by the force of necessity.

Then there is the unequal development of the two hemispheres of the brain, a most important and interesting subject.

The effects of ambidexterity upon the development of the two hemispheres of the brain, as elucidated by neurologists, have been well summarised by Mr. John Jackson * and others, and I need not dilate upon this part of the subject.

It is an old theory that the brain can act in a double capacity. So long ago as 1844 Dr. Wigan wrote a book † devoted to this subject.

He insisted that each hemisphere of the brain could act independently of the other. The evidences adduced by this writer, although very plausible, were not sufficient to prove absolutely convincing, and I cannot find that anyone since Dr. Wigan's time has brought forward any more practical evidence upon this point.

A short time ago I was much struck upon hearing Mr. Jackson remark incidentally that his daughter in practising writing with her left hand had also simultaneously written upon a different subject with her right.

This was so distinctly a case of dual action of the brain that I took steps to see the performance of this feat personally. In the presence of myself and a few medical friends the young lady wrote several words and two letters quite different in character simultaneously, one written with the right and the other with the left hand.

Then I tested her by getting her to read a paragraph in a newspaper while I kept her closely in conversation answering my questions given rapidly, questions which required her careful attention. She was then able to repeat almost word for word what she had been reading.

This seems to me to be the most conclusive proof that the brain (at least in this instance) is capable of entertaining two distinct currents of thought or to perform two cerebral actions simultaneously.

Whether this dual action is a faculty desirable to cultivate, or whether it militates against concentration of thought upon one subject, are questions upon which I am not prepared to offer an opinion; but the beneficial effect of cultivation of the use of the left hand equally with the right has been clearly proved.

These benefits are many, but I will here only refer to three of the most obvious:

1. The symmetrical development of the body.
2. The improvement in brain power generally.
3. The advantage of possessing two "dextrous" hands instead of one.

* The Mr. Jackson I refer to is the introducer of the upright style of penmanship as a hygienic method.

† Wigan (A. L.), 'The Duality of the Mind,' London.

A Contribution to the Study of Manubrial Dulness, especially as related to Enlarged Bronchial Glands.—H. B. Whitney states that the chief diagnostic importance of bronchial adenopathy lies in its differentiation from aneurysm or mediastinal growth. Pressure symptoms of adenopathy may be very pronounced. In one of the cases reported here there was recurrent paralysis and tracheal stenosis, and in two of the cases compression of the left bronchus. With such symptoms, diagnosis may at times be very difficult. Chief stress must be laid upon occupation and history. In the existence of manubrial dulness, the entire absence on the one hand of any sign of aneurysm, and on the other of any suggestion of malignancy, such as cachexia or enlarged cervical glands, should point to the careful investigation of the patient's antecedents and the condition of the lungs—especially with reference to pneumonoconiosis, such as slight consolidation or bronchitis. In most aneurysms, if there is any dulness it is to the right of the median line. The dulness of adenopathy is most often on the left. The dulness of early aneurysm is apt to be small, while that of adenopathy is more apt to be diffuse.—*Medical Record*, May 2nd, 1903.

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CLINICAL LECTURES

ON

DISEASES OF CHILDREN.

LECTURE XII.

THE RESPIRATORY DISEASES OF CHILDREN—*continued.*

Delivered at the London Hospital, December 2nd, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,

Assistant Physician to the Hospital, and to the Hospital for Sick Children, Great Ormond Street, W.C.

I NOW pass to the consideration of a disease which is extremely common in children, namely, *bronchitis*. Bronchitis is certainly commoner in childhood than at any other period of life, and the reason for that may be the tendency of children to get chilled owing to their large body surface; contributing causes certainly are rickets and the occurrence of teething. You constantly find bronchitis as one of those catarrhal manifestations of rickets of which I have already spoken, and you will find children who get an attack of bronchitis every time they cut a tooth. Why that should be I do not know. Some people say it is produced in some mysterious reflex fashion, but I think it is more likely that the teething simply lowers the child's resistance, like any other agency, and so renders it specially liable to suffer from chill. Now "bronchitis" in itself is never a sufficient diagnosis. That is true not only of children, but of grown-up people. But it is particularly true of children, because they are apt to suffer more than grown-up people from capillary bronchitis,—that is to say, inflammation of the small tubes, which may be a very serious matter. And so, having established the fact that the disease exists, you have to ask yourselves two questions: first, which set of tubes is involved, the large tubes or the small ones? and secondly, what stage has the bronchitis reached? is it early or is it late? With regard to the first of these questions, you will settle whether you are dealing with bronchitis of the large or of the small tubes by the character of the accompaniments. If the large tubes are involved you will hear low-pitched accompaniments—*i. e.* sonorous rhonchi—if it is in the dry stage, or more or less coarse bubbling sounds in the moist stage; whereas if the small tubes are involved the accompaniments will be high-pitched rhonchi if the bronchitis is dry, or fine crackling sounds if it is moist. Again, in a case in which the fine tubes are involved the accompaniments are far more numerous than in those cases where it is the large tubes which are chiefly affected. The obvious reason for this is that there are far more small tubes than big tubes in the lung, so inflammation of the small tubes will result in your being able to hear many accompaniments; and if you hear rhonchi coming from every quarter you can be certain you are dealing with inflammation of the small tubes. The other question is, what stage has the disease arrived at in your case? That is settled by the character of the accompaniments—whether they be dry or whether they be moist. In the early stage of the disease you have dry sounds, and in the later stage the sounds become both dry and moist, whereas in the latest stage the moist sounds predominate. The question of whether the large or the small tubes are involved in the inflammation is one of great importance in the prognosis, and that is chiefly why you wish to determine that point. You take a more serious view if the small tubes are chiefly involved, because the great thing you have to fear in a case of bronchitis in a child is that the condition may pass on to broncho-pneumonia—that is to say, to inflammation of the lung substance,—instead of being merely an inflammation of the lung tubes. The remote danger in bronchitis, you will remember, is that it leaves behind an unhealthy condition of the bronchial glands, which renders them liable to be the seat of tuberculosis. With regard to bronchitis, I would further say that you should always take a more serious view in a case in which the chest wall is very soft and yielding. Practically it amounts to this, that bronchitis in a rickety child is a far more dangerous affection than a similar degree of bronchitis in a child who is otherwise in good health. The rickety chest is so soft that it readily allows collapse of the lung, and the extension of the process to the collapsed lung, with the production of pneumonia.

The treatment of bronchitis must depend upon the stage at which the disease has arrived. If you are dealing with an early case where there is fever, more or less dyspnœa, rapid breathing, and high-pitched dry sounds, then you ought to put the child in a tent and let it have creosote inhalations. That is probably far more valuable than the

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administration of any amount of expectorants by the mouth. Here, at this hospital, in the out-patient room, you get a wrong idea of how to treat these cases; you cannot get out-patients to use bronchitis kettles, and so one has to fall back on the second best treatment—namely, the internal administration of expectorants. But when you have complete control of the case the best plan is to put the child in a tent and give it a bronchitis kettle, and use creosote as an inhalation along with the steam. It is well, also, that those cases should have either a cotton-wool jacket or a poultice over the back. It is a mistake to put poultices over the front of the chest, because they are too heavy; they increase the labour of breathing, and the child ought not to have anything to restrict the breathing. But a poultice over the back is often very valuable. You may use for the poultice linseed meal and mustard. There is no harm in giving, in the early stage, small doses of ipecacuanha by the mouth along with alkalies, such as citrate of potash or acetate of ammonia. And such children should be encouraged to drink large quantities of hot fluids, because there is nothing better for promoting the secretion from the bronchial tubes than the administration of hot fluids of all sorts. That is the meaning of the old domestic teas which used to be brewed. They acted not in virtue of the material in them, such as camomile, but simply in virtue of the fact that you are giving large quantities of hot liquid. In the later stage, when you have got to deal with much liquid mucus in the tubes, you will find it an advantage to administer an emetic, particularly where the child has difficulty in getting rid of the mucus and where there is much choking and a furred tongue. There are one or two conditions which contra-indicate the giving of emetics. One of these is the presence of much cyanosis. The reason is that in that condition the nervous system is, as it were, partially narcotised; it does not respond to the irritation of an emetic, and you are apt to have the ipecacuanha or other emetic retained, and great depression result without emesis. Great prostration is the other condition which contra-indicates the giving of an emetic in the second stage. In the last stage—that is to say, when the patient is convalescent—you should administer such things as iron and cod-liver oil, and apply stimulating liniments to the chest until all accompaniments have disappeared. You must make convalescence complete because of the great danger of tuberculosis following in the bronchial glands, which have become chronically inflamed.

I will speak next of *broncho-pneumonia*. This is a disease which you often see in the wards, one of very great fatality, so much so that about 17 per cent. of the total infantile mortality results from it. Broncho-pneumonia may be either primary or, more commonly, secondary, occurring in the course of another disease, such as measles or whooping-cough; and in such cases the broncho-pneumonia is very often the last straw, as it were; it is that which induces a fatal ending. Now your difficulty will always be, when you are in contact with a case of supposed broncho-pneumonia, to say when you are dealing with simple bronchitis and when the disease has passed on a stage further—namely, to broncho-pneumonia. I would like to emphasise the fact that physical signs will here be of little assistance to you; you cannot diagnose broncho-pneumonia with certainty from simple bronchitis by physical signs; you have always to consider the general symptoms. If there is high fever lasting more than two or three days, if there is much lividity and much depression, then you can be certain, no matter what the physical signs are, that you are dealing with the more serious affection—namely, broncho-pneumonia,—and not with mere bronchitis. If there is one physical sign of assistance more than another it is the development of fine crepitations towards the base; and if in any case of bronchitis there are fine crepitations towards the base of one lung, you should be suspicious that you have to do with patches of broncho-pneumonia. The patches of broncho-pneumonia may be so small, and so deep down, and so enclosed by healthy lung, that there may be no dulness at all, and you must not expect always to get dulness when you percuss such a chest. But the more lightly you percuss it the less likely you will be to miss such patches as exist, for you must remember that sometimes the patches are extremely thin; there may be only a little layer of broncho-pneumonia, and any heavy percussion will bring out the resonance of the underlying lung below that, and so you may miss the dulness. The other disease from which you may have a difficulty in distinguishing it is croupous pneumonia, and it will be well to put down in tabular form the points

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which should guide you in the distinction between croupous and broncho-pneumonia. Croupous pneumonia is always a primary disease, but broncho-pneumonia is very often secondary. Broncho-pneumonia is commonest in quite young children, in babies, whereas croupous pneumonia is not commonest in babies, but in children three to four years of age. Broncho-pneumonia usually comes on more or less gradually, and the previous health has often been bad; whereas croupous pneumonia comes on suddenly, and usually in a child who has before been perfectly well. Broncho-pneumonia usually affects both lungs, whereas croupous pneumonia is unilateral, affecting one lung or, much more likely, part only of one lung. In broncho-pneumonia the base of the lung is the part which is almost always involved, whereas in croupous pneumonia it is often apical. I do not know what proportion apical cases bear to basal ones; but the apical cases are very much commoner than you would think, and certainly more than they are in grown-up people. The course of broncho-pneumonia is ill-defined, and, instead of ending by an abrupt crisis, it ends usually by lysis. In croupous pneumonia, on the other hand, the course is well defined, and ends by crisis on the fifth or ninth day. The dulness in broncho-pneumonia is usually patchy and ill-defined, whereas in the croupous form it tends to follow sharply the outlines of the lobe. Another point of some value is that in broncho-pneumonia you get signs of general bronchitis, whereas in croupous pneumonia the rest of the lung seems healthy. These are the chief points to be attended to in making your diagnosis. The reason why it is so important to make the diagnosis is from the point of view of prognosis. Broncho-pneumonia is so fatal that about half the cases in hospital die, while, on the other hand, in croupous pneumonia recovery is the rule; deaths are very exceptional. Holt, in his statistics, found something like 4 per cent. as the mortality of croupous pneumonia in young children, which is, of course, far lower than in grown-up people. There are probably two reasons for the better prognosis in croupous pneumonia in young children. One of them is the great reserve power of the heart, so that you do not easily get paralysis of the right ventricle from over-distension. The other reason probably is that children have usually very healthy kidneys, and there is nothing, I suppose, except alcoholism, which often goes with it, which makes the prognosis in pneumonia more unfavourable than damaged kidneys.

The *treatment* of broncho-pneumonia is exactly the same as that of acute bronchitis, *plus* the administration of respiratory stimulants such as strychnine, and of general stimulants such as alcohol, if they are indicated; otherwise you do exactly as in acute bronchitis, keeping the child in a uniform temperature and using a tent and steam inhalations and counter-irritation to the back, and also giving, if you like, small doses of expectorants. In addition, as I say, if there be signs of respiratory failure you will use strychnine and alcohol, and you may require to employ inhalations of oxygen. The treatment of *croupous pneumonia* in children may be summed up in a word. There is no occasion to treat the case beyond putting the child to bed and generally nursing it. I usually treat them as out-patients unless they are severe cases, and tell the mothers to take them home and keep them in bed, and sponge them if they are hot and restless. You can also, if you like, give them a diaphoretic mixture.

I ought to say a word about the *diagnosis of croupous pneumonia* from other conditions. You will be very apt to mistake it for acute gastritis. If you get a child with acute vomiting and a furred tongue, do not conclude too quickly that you are dealing with a case of gastritis; you should remember the possibility that it may be croupous pneumonia, because the physical signs of croupous pneumonia, particularly when it affects the apex of the lungs, are often very hard to make out. The child may be near the end of its illness before you are able to make out any dulness or bronchial breathing; you have to diagnose it from the mode of onset, the high temperature, and, above all, from the rapid respiration; the physical signs, just as in broncho-pneumonia, take a secondary place.

Collapse of part of the lung, or "atelectasis," as it is sometimes termed, is a condition which you will meet with not uncommonly in children. It is sometimes *congenital*—a persistence, in part of the lung, of the fœtal condition, and is here associated with the symptoms spoken of as asphyxia neonatorum. Frequently, however, it is *acquired*, coming on in the course of other pulmonary diseases. Softness of the chest wall greatly facilitates its pro-

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duction; hence atelectasis is more frequently met with in rickety children than in others. Anything which compresses the lung, *e. g.* an effusion into the pleura or pericardium, may induce it, and it is stated to result also from blocking of the smallest bronchioles, either from the accumulation of secretion or from swelling of their lining membrane. This may explain the common occurrence of areas of collapse in the course of a broncho-pneumonia.

You will find the physical signs of collapsed lung rather puzzling. If extensive, the note over the affected area will be impaired and the breath-sounds feeble or even slightly bronchial in character. Occasionally, also, a few moist sounds may be heard. The general symptoms, as you can readily imagine, will vary with the amount of pulmonary tissue involved. In slight cases they may be almost absent, but if the collapse is extensive there will be a greater or less degree of cyanosis, and you will notice that the child breathes and cries feebly and that the extremities are apt to be cold.

In the *treatment* of this affection you must aim at promoting efforts at inspiration, so as to re-inflate the collapsed areas. This can best be done by sprinkling cold water on the chest and by friction with stimulating liniments. Some writers advocate vigorous spanking, repeated, if necessary, several times in the day; and though such treatment may sound brutal, it is certainly rational, for crying necessitates deep inspiration.

Pleural effusions are not uncommon in children, and are apt to be overlooked. Here, again, physical signs are of doubtful value. There will be dulness over the effusion, but this need not be very intense; and not uncommonly, owing probably to the great conductivity of the child's chest, which I have already spoken of, the breath-sounds are preserved over the affected area, and may even have a feebly bronchial character. Hence, as you will readily understand, pleural effusion is not always easy to diagnose from consolidated lung, and sometimes you can only make sure of its existence by exploration. You need have the less hesitation in having recourse to this aid if you remember that in any case it is practically impossible to decide between clear fluid and pus without the use of the needle. The general symptoms will not help you, for clear effusion is often attended by a greater degree of fever than empyema, and, indeed, the symptoms of the latter in young children are often curiously slight. Even the presence or absence of a leucocytosis is no certain guide. It may be well for you to remember, however, that below the age of three clear effusions are rare, whereas empyema is comparatively common.

The *treatment* of pleural effusion I need not describe, as it must be conducted on exactly the same lines as in grown-up people.

In a previous lecture I have already referred to *pulmonary tuberculosis* in children, but I should like to emphasise again the fact that phthisis, as one sees it in the adult, is very rare in early life. In young children, as we have already seen, pulmonary tuberculosis usually starts in the bronchial glands, and invades the lung secondarily. I would also remind you once more of the great difficulty of diagnosing between some forms of tuberculosis in the lung and chronic broncho-pneumonia; over and over again you will be likely to make mistakes in this matter, for the physical signs of these two affections may be identical.

The *fibroid* form of tubercle is not uncommon even in young children, but here, again, you must remember that pulmonary fibrosis consequent upon an empyema or an imperfectly resolved broncho-pneumonia, or even upon collapse, is also frequently met with. Owing to the absence of sputum, too, your difficulties are rendered greater than in the adult, for you are deprived of that aid to diagnosis, and often you can only come to a correct conclusion by a prolonged study of the case.

Menstruation and the Corpus Luteum.—Dr. O. Th. Lindenthal ('Wiener klinische Wochenschrift,' March 12th) noticed during an ovariotomy, bleeding from a fresh follicle which had been ruptured by the examination preceding the operation. The patient, who had heretofore always menstruated regularly, then had an atypical menstruation. The author suggests that the amenorrhœa frequently following a unilateral ovariotomy may be due to the fact that the extirpated ovary contained the follicle which would have given the impulse to the menstrual flow. The ovaries appear to possess an alternating function, and the removal of one ovary is not always followed by the assumption of a vicarious function by the remaining one. —*New York Med. Journ.*, May 2nd, 1903.

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A CLINICAL LECTURE

ON

"A CASE OF DISPLACEMENT OF BOTH TESTES IN PERINEO, WITH AN INGUINO-PERINEAL HERNIA ON THE RIGHT SIDE."

Delivered at Guy's Hospital.

By R. CLEMENT LUCAS, M.B., B.S., F.R.C.S.,

Senior Surgeon to and Lecturer on Surgery at the Hospital.

GENTLEMEN,—I have for consideration to-day a case of displacement of both testes into the perineum, associated with an inguino-perineal hernia on the right side. I will first read to you the details of the case, and then I will comment upon it, and consider the question of the normal and abnormal descent of the testes, and the causes which determine these abnormal conditions.

A little boy,* three years of age, was admitted to a cot in Lazarus Ward on May 2nd of last year, on account of a swelling in his right groin and perineum. The boy was a fairly healthy son of somewhat unobservant parents. They had apparently noticed nothing amiss with him during his infancy, and, except for slight troubles when teething, his general health had been uniformly good. One day the boy was trying to climb on a chair, when he suddenly began to cry and put his hand on his right groin. His mother looked and found a swelling there. She took him to a doctor, who said he had a rupture and a misplaced testis, and advised her to take him to a hospital. When admitted he was found to be suffering from a hernia which extended from the inguinal canal past the outer side of the scrotum into the perineum behind this pouch. It was somewhat tender but reducible. At the back of this swelling the

* This case was read before the Society for the Study of Disease in Children on June 21st, 1902.

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right testis could be felt fixed in the perineum. On the left side there was no hernia, but a swelling was evident just behind the left half of the scrotum, and this, when grasped, could be felt to be the left testis. The penis was well developed, but the scrotum was small, puckered, and empty. Physical examination thus clearly proved that the case was one of double testis perinealis which had been overlooked until a hernia descending into a congenital sac on the right side drew attention to the congenital displacement. An operation was undertaken for the cure of the hernia on the right side. On May 14th, A.C.E. mixture was administered, and an incision was made over the external abdominal ring. The cord was exposed and the testis drawn up. It was found attached by a strong gubernaculum to the tissues of the perineum, which band required division before the organ could be raised. The congenital sac of peritoneum was then isolated and excised from the internal abdominal ring to the top of the testis. The lower portion was sewn up by a fine silk suture to form a tunica vaginalis, whilst the upper end was transfixed, ligatured, and tied to the abdominal muscles. The right side of the scrotum, after being stretched with the finger, was invaginated, and two small silk sutures were used to fix the testis to the bottom of the scrotum. Next the inguinal canal and the external abdominal ring were narrowed and strengthened by cross silk sutures cut short and buried, and finally the skin was sewn up with a horse-hair continuous suture, and the wound sealed with collodion and iodoform. No drainage was employed, and no incision made into the perineum. The operation before the days of asepsis used to be an exceedingly fatal one in these cases where the testis was displaced into the perineum, and it was thought to be always necessary to have an opening into the perineum for drainage. That opening naturally got infected, and as it was not the custom to close the opening of the peritoneal cavity, the infection spread to the peritoneum, and the cases were nearly always fatal. The wound in our case healed primarily, and the child had a normal temperature for six days, after which his temperature suddenly ran up; and a few days later he developed the rash and coryza of measles, and had accordingly to be sent to the isolation ward. The measles ran a normal course, and in no way interfered with the proper union of the parts severed in the operation. He was discharged from the hospital on June 12th convalescent from the measles, and with the wound soundly healed. On June 19th he was brought again to the hospital with the object of having the left testis implanted in the scrotum. On examining him, however, it was found that no further operation was necessary. As the result apparently of the double spica bandage carried from thigh to thigh and across the perineum to retain the dressings in place after the operation

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on the right testis, the left was now found to be in its proper position in the left side of the scrotum. The scrotum had undergone development, and the testis showed no tendency to escape again from the purse. That is an interesting case, the details of which I have endeavoured to depict on the blackboard. On the right side the testis is behind the scrotum, and there is an open vaginal process of peritoneum filled with a hernia. On the left side the testis could be felt and seen behind the left half of the scrotum. We operated upon the hernia, replaced the right testis into the scrotum, and the effect of the bandage was, as I have said, ultimately to push the left testis back into its proper position in the scrotum. The latter could not have been attached by a strong gubernaculum as on the right side. It might perhaps be argued that the effect of replacing the right testis in the scrotum might have stimulated the development of the fibres of the gubernaculum normally attached to the left side of the scrotum, and so have led to the transplantation of the left testis into the scrotum; but I am more inclined to think it was a mechanical replacement caused by bandaging across the perineum. You will see the condition depicted in this diagram. I have also had some diagrams put up showing imperfect descent of the testes.

I will now ask your somewhat careful attention to a few remarks upon the development and descent of the testis. We will endeavour to explain the ectopia testis, as that condition is described, when the testis, instead of passing into the scrotum, passes into one of these abnormal positions, such as into the perineum, on to the pubes, or over Scarpa's triangle. First of all let me remind you that in the lower vertebrates—birds, reptiles, amphibia, and fishes—the testes are throughout life abdominal organs. When we reach the lower mammals they still remain within the abdomen. The Monotremes, of which the Ornithorhynchus is best known, have testes within the abdomen, and these testes never come to the surface. The elephant and the rhinoceros also have intra-abdominal testes. Then you meet with a certain number of animals in which the testes appear outside the abdomen at periods of rutting, whilst at other times, when the animal is not rutting, the testes are retained within the abdomen. This is a to-and-fro arrangement which is met with in many insectivora and rodentia. The hedgehog is an exceedingly good example of this, and it has been very carefully studied by Dr. Joseph Griffiths, of Cambridge.* The hedgehog in summer has well-developed testes lying outside the abdominal cavity, but in winter these organs recede into the abdomen, and a microscopical examination shows that their structure under these two conditions is totally different. The testes when outside the abdomen, at the rutting season, have well-developed tubules; the tubules are double the size that they are in winter, and there is little intertubular cellular tissue. They are lined by a layer of cubical epithelium, followed by layers, two or three in number, of cells in which the nuclei can be seen to be dividing. And finally you notice on the inner side tufts of spermatozoa coming from the cells, and a wide lumen in which there are free spermatozoa and coagulable secretion. On the other hand, in winter the tubules are about half the size, the tunica propria of the tubules is thicker than in the summer, the intertubular cellular tissue is increased in amount, there is no lumen in the tubes, they have a layer of cubical epithelium within the tunica propria, and the lumen is filled up with polygonal cells with large round nuclei in which no traces of spermatozoa can be detected. There is no spermatogenesis going on in the winter time in the hedgehog's testes, and it is only in summer that the testicles develop and the tubules double in size. The testes come out of the abdomen into a prominent position, and at that time you find within the tubules these layers of cells with a well-developed lumen, and spermatozoa in various stages of development.

Now the testes in man are developed within the abdomen and pass down through the inguinal canals into the scrotum. I shall endeavour to trace the development of the testes and their descent into the scrotum. In a certain number of cases one or both testes may remain within the abdomen, and it is almost certain that if not in every case, in almost every case, a testis which does not pass from the abdominal cavity into the scrotum does not undergo its proper and full development. Just as the hedgehog's testis during winter is an abdominal organ, showing no spermatogenesis, so the human testis which fails to make its proper

* 'Journal of Anatomy and Physiology,' vol. xxviii, p. 209.

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descent into the scrotum is an imperfectly developed organ, showing defects in the development of its cells, showing small tubules with thickened tunica propria, and no signs whatever of spermatozoa. One testis, however, is quite sufficient for all developmental purposes, and also for procreation. These undescended testes, supposing neither one nor the other to make its appearance in its proper position, nevertheless have a certain influence on the developing organism. For those persons in whom both testes are retained in the abdomen, who are spoken of as cryptorchids, show no sign of that characteristic development, or arrest of development, which is peculiar to the eunuch. If the testes be removed in early infancy, then you get such a change produced in the organism that the boy never develops into a properly formed man. He is stunted in growth, he has a wide pelvis, he has a fleshy, soft exterior with a tendency to the development of the mammary glands, and he has a high-pitched voice like a woman's, and grows no beard. But these cryptorchids develop as men, having their proper manly growth; they have narrow pelves, they have deep voices, and they grow beards like men. But still there must be a very wide difference between a properly developed man and a cryptorchid. It is extremely doubtful whether in any case where both testes fail to pass out from the abdomen into their proper situations,—whether such an individual, though he have a man's growth and narrow pelvis, a man's voice, and may grow a man's beard, can procreate. Microscopical examination of retained testes is against it; it shows, as I have said before, that there is a lack of development in these organs, that the tubules are small in size, thick as regards their tunica propria, that their intertubular tissue is increased in amount, and that the cells in their interior show no signs of spermatogenesis. In many cases these cells have been found, according to Dr. J. Griffiths, to be columnar, forming a single layer; and in some cases this layer of columnar cells is almost entirely absent or defective. I have tried to contrast the eunuch with the cryptorchid. In neither of these, apparently, can procreation take place; yet, you see, a cryptorchid is quite a different individual to an eunuch. As regards his appearance, no one would judge that there is anything in the cryptorchid different to an ordinary man, if no examination be made with regard to his generative organs. More than that, it seems that in a very large number of these cases it is possible that such an individual may marry and may have proper connection, and may have an emission of secretion—I will not say of semen. The secretion in such a case is probably from the prostate, the vesiculæ seminales, and from Cowper's glands, and it shows no signs of the presence of spermatozoa. I may say that the sterility of the cryptorchid has been contested by certain writers, among others by a late colleague of mine, who, I believe, collected three cases in which he showed that three married men, who were cryptorchids, nevertheless had children. But you see, this is always open to doubt. It may be certain that a child is the child of its mother, but it may not be absolutely certain that the child is the child of its reputed father. So here comes in the possible fallacy. The microscopical examination of retained testes is against that conclusion.

I will now go on to say a few words about development of the testes and the descent of testes into the scrotum. You will remember that in early fœtal life at the back part of the abdominal cavity is developed an organ with transverse tubules, and a duct known as the Wolffian body, the duct passing down to the cloaca. This Wolffian body serves the purpose of a temporary kidney, and close in connection with it is developed a mass of epithelial cells called the germinal epithelium, which forms a little ridge called the germinal ridge, which afterwards becomes either the testis or the ovary. To-day we have only to do with the testis. I want you to understand that this mass of epithelial cells, which afterwards becomes a tubular organ, lies close in contact with this Wolffian body at the back part of the abdominal cavity; and as it is developed it is wrapped and tied to the back by a fold of peritoneum, and held in position by what is called the mesorchium. This Wolffian duct that I have spoken of, the duct of this temporary kidney, forms one part of what we are apt to speak of as testis when we are describing that organ in the dissecting room. The Wolffian duct becomes joined on to this germinal mass, and forms the vas deferens and the epididymis. Part of the Wolffian body, or part of the tubules of the Wolffian body, form the vasi efferentia and the coni vasculosi and the top of the globus major. And so, you see, the

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testis and the epididymis are derived from different sources, and the epididymis does not always join on to the testes. In an undescended and undeveloped testis that I removed some years ago when performing an operation for the radical cure of hernia, I found that the vas deferens terminated in a kind of vascular area, and did not actually join the testis. In most of these ill-developed testes which you find in the inguinal canal and thereabouts, there is a wide separation between the epididymis and the testis, and in some cases, as in the one related, it is quite certain the epididymis does not join the testis at all. Now beneath the testis, where it is held at the back part of the abdomen by its mesorchium, and running down from that, you observe a band of unstriped muscular fibres which is known as the gubernaculum testis. This gubernaculum testis is connected with the bottom of the testis, and spreads out below, some of its fibres also being attached to the peritoneum in front of it. It is generally believed (though it has been disputed by at least one very able anatomist, Dr. Cleland) that the gubernaculum is the main structure which causes the descent of the testis into the scrotum; and that view I believe to be the correct one. This band, which I have endeavoured to represent diagrammatically, descends from the testis, along the inguinal canal, and sends one external branch to be attached to Poupart's ligament inside the abdominal wall. Another branch comes through the external abdominal ring and gets attached to the front of the pubes, whilst a third band is attached to the bottom of the scrotum. The contraction of these muscular fibres is believed to determine not only the descent of the testis, but also the descent of that tube of peritoneum, known as the vaginal process of the peritoneum, which precedes the descent of the testis into the scrotum. Some of the fibres of the gubernaculum in front are attached to the peritoneum, and it is believed that they influence the descent of the tube of peritoneum into the scrotum. The band, then, in the inguinal canal attached to Poupart's ligament tends to draw the testis through the internal abdominal ring into the canal. The one attached to the pubes would tend to draw it out from the external abdominal ring; and the main branch should draw it into the scrotum. As it descends, the testis gets wrapped round by these muscular fibres, and those inverted muscular fibres form part of the cremaster muscle; other fibres of the cremaster muscle are apparently derived from the edge of the internal oblique muscle. That is the normal descent.

To-day we have to do with an ectopia testis, or one displaced into the perineum. The fibres attached to the scrotum and the fibres attached to Poupart's ligament and the pubes are not the only fibres belonging to the gubernaculum. By careful dissections it has been found that fibres belonging to the gubernaculum spread out in various directions; some to the perineum, and of these some even reach the sphincter ani, the ramus of the ischium, and the perineum beside the anus; while others have been found spreading out in the neighbourhood of the saphenous opening in Scarpa's triangle, and others round the ring towards the anterior superior spine of the ilium.

There are various forms of ectopia besides the one which I have shown here. In some cases the testis has been found on the pubes, and that would be explained by the fibres belonging to the pubic band being stronger than the others, and drawing the testis to the root of the penis or on to the pubes. In other cases the testis has been found in Scarpa's triangle, and it has been thought to have come out from the crural canal; but there is no evidence available of any testis having come out through the crural canal. It is simply that some of the fibres of the gubernaculum which appear and disappear in fœtal life have drawn the testis to the position of the saphenous opening. It has been found also in the fourth situation to which I have referred—that is to say, lying on the external oblique towards the anterior superior spine,—and some of the fibres of the gubernaculum have been traced in that direction. So there are these four situations in which you may find a testicle which has passed through the inguinal canal but yet has not passed, as it should do, into the scrotum. The most common form is that on which I am lecturing to-day,—that is to say, when it is lodged in the perineum. The next in frequency is that when it is lodged on the pubes; the next when it is in Scarpa's triangle; and the least frequent is on the external oblique, towards the anterior superior spine.

The date at which the testis descends, or when it commences its descent, is at the end of the fifth month. At the sixth month it lies in the iliac

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fossa, and at the seventh month it enters the inguinal canal. At the eighth month, according to most authorities, it should have descended into the scrotum. Some are inclined to put it a little later, and do not think it is normally at the bottom of the scrotum until towards the end of the ninth month,—that is to say, not very long before birth. Ordinarily, if you find a normal fœtus born with both testes in the inguinal canal you will be fairly safe in saying that it is about a seven-month fœtus. Professor Cunningham, amongst others, is rather inclined to put the descent of the testes a little later than I have mentioned. It is accompanied, or slightly preceded, by the vaginal process of the peritoneum; and in almost every case where the testis is ill-developed and does not descend properly into the scrotum it is associated with an open vaginal process of peritoneum. Normally this process should close at the internal abdominal ring before birth, and a contraction in the sac takes place just above the testes at about the same time that the closure is taking place above; and those two portions should be united by the closure of the canal all the way through, so that normally about ten days or a fortnight after birth the vaginal process of the peritoneum should be only represented by the tunica vaginalis and a fibrous cord extending from the internal abdominal ring to the top of the testis. In our particular case, on the right side the process of peritoneum remained open, and it was complicated by a hernia, which we operated upon. On the left side we may presume that it has closed; at any rate there was no evidence of hernia on that side, but that alone is not complete evidence of closure. It is possible that the vaginal process of peritoneum may be open on the left side and no hernia at present have come down into it; because we find in many cases a hernia may not appear in infancy, though it may suddenly come down at the age of puberty or afterwards,—so suddenly that it could not be from a gradual stretching of the peritoneum, and must have been a hernia in a congenital sac. In the human subject the vaginal process of the peritoneum closes if the process is carried on to completion, and we are peculiar in this respect—that we are the only animal in which the tunica vaginalis is cut off from the rest of the peritoneal cavity. Even the anthropoid apes have all open tunicæ vaginales, and therefore they are liable to congenital herniæ. It is one of the characteristics of man, apparently associated with his erect position, that the tunica vaginalis becomes cut off completely from the general peritoneal cavity when the process is carried on to perfection. That is a thing to remember and to be proud of.

I will now make a few remarks upon the operation which was undertaken to put the testis back into the scrotum and to cure the hernia. The radical cure of hernia in infancy is by no means an easy operation. The reason it is so difficult to perform is that the tissues are so delicate, and the vas deferens is so difficult to isolate, that it is very easy indeed to sacrifice the future usefulness of the testis when performing the radical cure of hernia. In early cases of this nature you are always operating upon an open vaginal process of peritoneum. It is not an acquired hernia, nor is it in many cases a funicular hernia cut off at the top of the testis from above. You have a big open tube running from the abdominal cavity and surrounding the testis. To carry out a radical cure it is necessary to destroy this tube and close the upper opening. We dissected it carefully out and isolated it from the vas deferens. We made a tunica vaginalis below and cut away the rest, closing the upper opening which communicated with the abdominal cavity. We then proceeded to place the testis in the scrotum. In order to accommodate the scrotum for receiving the testis I pushed a finger through the wound into the scrotum and stretched it well to make a good cavity. Then we proceeded to draw on the cord and testis, but before we could raise it we had to cut through a strong band attaching it to the perineum. We did all this through the same wound, and we were then able to isolate the testis and put it into the scrotum. To fix it into the scrotum we first invaginated the scrotum from below by pushing the finger in from the outside, and then put a couple of stitches through the tissues forming the scrotum from the inside, and sewed the bottom of the scrotum on to the piece of gubernaculum which was at the bottom of the epididymis. In that way we sewed the testis into the bottom of the scrotum so that it could not escape from it. We next passed ligatures across the inguinal canal and tightened it up, leaving a space for the cord to pass along. We narrowed the ring, which was large, and then closed the wound. The stitches with

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which we sewed the testis down to the scrotum were left buried in the scrotum, and they caused no irritation there.

An interesting circumstance was that when we anticipated that we should have to do an operation on the other side, and the patient came up again for that purpose, we found that the testis on the left side had been replaced, during the convalescence, into the scrotum on that side. In whatever way this took place it must, I think, be attributed to the operation which we performed on the right side, and probably to the mechanical pressure we put upon the perineum when bandaging on our dressings. It is possible, I would say, that the stimulating effect of the testis being put into the scrotum on the right side might have brought about development and stimulation of these fibres of the gubernaculum which were attached to the scrotum on the other side; and in that way there might have been a pull from the perineum towards the scrotum on that side. That is one view which I think may be taken of this replacement. I am rather inclined myself to favour the view that the main force in bringing this testis back on the left side to its proper position in the scrotum was the bandaging across the perineum, which was carried out very carefully by my dressers in order to prevent any infection of the wound from the rectum.

Now comes the question, what will be the future of this boy? Is he to be one of those who, like cryptorchids, may have a fully developed masculine frame without any chance of procreation? I am inclined to predict for this boy a future similar to that of any normally developed man. In the first place, it has, I think, been shown by others that testes which have passed down into the perineum show better development than those which are arrested halfway. It is rather that it has gone through its course and just missed its proper position, than that an early defect in development prevented it from descending.

There have been many views with regard to non-descent, but I think none so worthy of being recorded as that of our great surgeon John Hunter, who suggested many years ago, without any microscopical examination whatever, that the reason why the testis failed to find its way into the scrotum was that it was, from its earliest time, defective in development, and that in consequence of its defective development it failed to stimulate those other forces, such as the gubernaculum, which should, under normal circumstances, have carried it down into the scrotum. One more word for this great surgeon, John Hunter. It was shown by my esteemed friend Dr. Arthur Davis, in a learned and thoughtful Hunterian oration given last year,* that John Hunter not only indicated from his observations the cause of the non-descent of testes, but he also pointed out that those in whom the testes were undescended, though unable to procreate as he believed, nevertheless for some reason or other put on the normal form of man, and did not resemble eunuchs; and he it was who first suggested that this organ, besides secreting semen, had an internal secretion which so extraordinarily affected the development of the whole body as to bring about the normal masculine form. That is now one of the best received doctrines of the present day. The question of glandular therapy has developed in a marvellous way during the last few years; and all this was foreshadowed by that great and thoughtful surgeon, John Hunter, more than a hundred years ago.

ON ABDOMINAL TUMOURS,

THE NATURE OF WHICH MAY BE DETERMINED BY BIMANUAL EXAMINATION.

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GENTLEMEN,—It may be well, as a preliminary step, to define what is meant by bimanual examination, and to say something as to the best method of carrying this out. That this is not totally unnecessary I believe from my own observation. Many men in general practice I find are shy of performing it at all, more especially in unmarried women, where it is often necessary; and when they do attempt to carry out this manœuvre they do it in a way that does not afford them the full amount of information which it is possible to obtain. Standing on the right side of the patient, they introduce their right forefinger into the vagina

* The Hunterian Oration on "Organotherapy," 'Brit. Med. Journ.,' April 19th, 1902.

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and place their left hand on the patient's abdomen from the side above the thigh. By so doing, the sensitive pulp of the internal finger is carried backwards against the sacrum, where but little is to be felt; nor can the examiner, except by a most awkward twist of the forearm, turn the sentient portion forward so as to oppose it to the fingers of the opposite hand. Very quickly the hand in this position becomes tired and exhausted, and the finger is withdrawn long before the parts have become supple enough for the examiner to feel anything which lies between.

It cannot be too strongly insisted upon that patience and perseverance are indispensable in order to obtain any satisfactory result. When the palpating external hand is laid upon the abdomen, it finds, in nine cases out of ten, a resistant surface which feels like stiff cardboard beneath the skin. If the examiner's hand is warm and soft, and is gently but firmly applied, the cardboard sensation gradually changes to one of thick blanketing, which becomes progressively thinner until at last the feeling is that of a thin supple sheet of linen covering the internal organs. But this must be waited for. Any attempt to hurry matters by digging in the tips of the fingers only brings back again the earlier stages of resistance, and it is evident, therefore, that both patient and examiner must be in such a position that waiting is not synonymous with exhaustion, since the real examination can only begin when the last stage is reached. If the fingers have by that time lost their power of accurate observation, the examination must necessarily be perfunctory, inefficient, and without adequate result.

Occasionally, especially in women who have borne many children, the abdominal wall will be much more lax and easy to manipulate. Any one beginning bimanual examination with such a case will obtain a very false idea of the real difficulties which often present themselves; very soon he will find himself confronted by another, in which such an examination as he has been able to make in the first will appear impossible. His range of observation will be most unnecessarily restricted if he confines it simply to the first class, and the detection of morbid growths in young women, and especially in men, will be for him mere guess-work.

Bimanual examination is indeed by many thought of as vagino-abdominal alone, and therefore necessarily only possible in women, and only in those who are married. Those who practise it most frequently, however, find that the rectum in both sexes is the more convenient and satisfactory canal. It should, of course, be previously emptied, preferably by enema. When empty, it has two distinct advantages. First, it is the most posterior, and the finger in it finds all the objects which require examination between it and the palpating hand on the abdomen. Second, the finger in the rectum can penetrate much further than in the vagina, not being blocked as it is in the latter canal by the fornices.

The best position in which a patient can be placed for the performance of bimanual examination is the left lateral, which differs from Sims' position in that the legs are placed parallel one above the other and slightly bent, not with the right knee crossing over the left thigh, as in the position described by that authority. If the thighs are so placed, one directly above the other, the pelvis is made to lie square with the bed or couch surface, which is not the case if the upper knee is allowed to project beyond the other; and the introduction and use of the abdominal hand is thus rendered much more easy and effective.

The patient, lying on her left side, is brought to the edge of the couch or bed, with the hips parallel to the right side of the latter; the body is then flexed upon the thighs until it forms a right angle with them, and the head lies on the left side of the bed furthest from the examiner. His *left* forefinger, properly oiled, and having the nail well pared, so that it can in no way injure the delicate lining membrane of that canal, is passed slowly and gently into the rectum. When once it has penetrated the sphincter it should be pressed backward towards the coccyx, when the muscle will be felt to yield, and more room will be obtained. Always slowly and gently, the finger is made to penetrate more and more deeply. If it comes into contact with one of the rectal valves or folds of mucous membrane, a very little deviation of its direction will enable it to avoid the obstacle. When it has been introduced to its fullest extent the right hand is passed between the thighs and made to lie flat upon the abdominal wall. A pause should be made here, in order to allow the muscles of that wall to subside from their first instinctive

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contraction, and then, very slowly and gradually, the two hands should seek to approximate one another. When the abdominal wall has reached the last stage described above, the fingers of the external hand should search for and touch the tip of the internal finger. Any attempt to do this at first would be futile, and, by exciting recontraction of the abdominal wall, not only defeat its own object, but indefinitely prolong the process—causing, moreover, distress to the patient and loss of defining power to the hands of the examiner.

Usually the external hand will find the internal first just above the pubes, and this external hand will often by beginners be found to have been placed too high, and require to be brought down nearer the pubes. Any change in position of this, the external hand, must be made gradually, and by a sliding movement. If the hand is raised and re-applied, all the stages will have to be gone through again. When once the fingers touch they should be kept in relation with one another from that time onwards until the conclusion of the examination.

The main landmark which is to be sought internally is, in the female, the uterus; in the male, the prostate gland. Both are solid bodies with well-defined relations topographically with other viscera, so that if once these have been recognised, and their outlines mapped out, we have at once a clear idea of the position of any other swelling.

As the two sexes differ materially in the nature of most of the swellings which are probable or possible of occurrence in them, it will be well from this point to consider them separately, and as the male has the fewest we will consider them first.

Having found the prostate, the first thing is to determine its own condition. It may be extremely tender, as in **acute prostatitis**. Whilst the general mass may be firm, at one point softening and elasticity may be felt, as in **prostatic abscess**. The whole organ may be enlarged, and of wooden consistence, as in **chronic prostatitis**. Differing from this in consistence, and in a much more irregular outline, is the ordinary **enlarged prostate** of elderly men, which may be well defined in many cases by recto-bimanual examination, or still better with the aid of a vesical sound in the bladder and the finger in the rectum. By this means, without operation, a clear idea may frequently be obtained as to whether the enlargement is in the median or lateral lobes, or is simply a urethral fibroid collar. **Impacted calculi** behind an enlarged median lobe can also be detected.

Some portion of the prostate may be nodular, and these nodules stony hard, as in **prostatic calculus**, or **prostatic carcinoma**. Inasmuch as prostatic calculus is usually formed in one of its glandular tubules which open upon the urethra, the passage of a sound which produces a grating sensation as it passes over the projecting surface of the stone will often serve to differentiate these two. But in all these conditions, as in all others, the true appreciation of the differences between them is a matter of practice, and the tyro will find it a great help to have, by embracing every opportunity of palpating normal prostates, a clear standard of what the organ is like under normal conditions, and so be able to appreciate any deviation from this.

Posteriorly to the prostate, and on one or other side, the swollen vesiculæ seminales may be felt. Normally, these are elastic and vermiform. In **tubercular** disease they may be rugged, swollen, with small nodules interspersed amongst them. Immediately above and outside these a hard mass, stony hard, may be felt, slightly movable sideways but seldom upwards—a **calculus** wedged **in the vesical end of the ureter.**

Passing from the prostate and exploring the space in front of it, especially in that part which is most dependent, which in this position will be the left side, the presence of **a calculus or other foreign body in the bladder** itself may be made out. Such a body will be, unless impacted in the ureteral orifice or behind an enlarged prostate, movable with fair ease, and will follow alterations in the position of the patient, always finding the most dependent situation.

But it is in the female that most swellings in this region occur, and some which are common to both sexes, such as tumours springing from the pelvic walls themselves, being found most frequently in women, it is convenient to describe them together with those peculiar to that sex.

Here, again, after reducing the enclosing walls to a properly supple condition, we seek first for the main landmark, which in women is the uterus.

And here, again, scarcely too much stress can be laid upon the necessity of examination, whenever practicable, of the normal uterus, so that the examiner may have in his mind a reliable standard

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of the shape, size, and consistence of that organ in its four normal states. The same uterus differs in all three of these characters according as it is virgin, pregnant, parous, and senile; although all these are perfectly normal states, and have to be differentiated from such conditions as metritis, subinvolution, pyometria, or fibroma. Only persistent practice can enable an examiner to appreciate fully these differences.

The uterus itself may be the seat of abnormal swellings. The first point which the finger impinges upon is the uterine os, and passing upwards from this, as a rounded firm pillar, is the cervix. Felt from the rectum it is much more prominent than would have been expected, and may easily be mistaken for the entire uterus. If, then, the **fundus is retroflexed**, this portion of the uterus will feel from the rectum as a solid rounded growth or tumour, lying behind the upper part of the cervix. This sensation may be corrected by examination through the vagina; but the mistake may be suspected if the fingers of the abdominal hand, made to meet the rectal finger below and in front of the os, feel no firm rounded body in front where the fundus should normally lie. By tracing the cervix upwards with the rectal finger, whilst at the same time pushing upwards the rounded mass behind, the angle between the two, which at first appeared to mark their separation, will open out, and as the fundus reassumes its normal position the two will be found to be continuous; whilst the abdominal hand in front may recognise the upper convex surface and, grasping it, convey its movements to the cervix beneath, the finger touching which will then recognise the fact that such movement is equally common to both.

But the uterine fundus may be demonstrably in front in its normal position, and yet a mass may be present behind and between the solid uterus and the rectal wall. This mass may be indefinite in shape, possessing no definite boundary lines, boggy in consistence, and spreading from side to side. It is a **hæmatocele**, and is practically always associated with ectopic pregnancy; the history and other signs not within the scope of this paper will be present; or, on the other hand, the boundaries of the mass may be definite. If it is hard and rounded it may be a **pedunculated subserous fibroid**, in which case it will not be tender, and probably will show some secondary bossing of its surface, whilst there will be no accompanying free fluid in the peritoneum. Or it may be an **ovarian fibroma**, in which case the surface will be entirely smooth, and some free fluid will be present. The hardness of both these is not stony, unless, indeed, the fibroma has undergone calcareous degeneration, in which case it will probably not be the only mass of the kind to be felt attached to, or incorporated in, the uterine substance. The swelling may not be as hard as a fibroma, yet firm and rounded with well-defined outline, and giving a peculiar sickening sensation to the patient when pressed upon. Such pressure will also make it spring away out of reach, only to return again when the pressure is relaxed. This will be a **prolapsed ovary** which has fallen into Douglas's pouch. The same swelling may be intensely tender, and not so freely movable, in which case the **ovary** is probably **inflamed**, and more or less adherent. But the swelling in this situation, whilst possessing somewhat definite outlines, may be soft and elastic; whilst the surface is rounded it is not circular, but gives the sensation rather of a coil of small intestine. If it moves easily out of the way under light pressure, it may indeed be such a coil, but if it moves very sluggishly, or not at all, it is more probably a **distended Fallopian tube**. Whether this is distended by retained secretion, by pus, or by blood, the history of the case will probably determine. It is readily distinguishable from free blood in Douglas's pouch by the fact that the outlines of the tube can be recognised.

Passing from the space behind the uterus to that organ itself, we may find it in one of several conditions. It may be generally and evenly enlarged, as in pregnancy which may be normal, or hydatidiform, or it may be in a condition of general fibroid degeneration. For diagnosis of purely uterine enlargements it is best to place the internal finger in the vagina, as many points are only to be appreciated in this way. Thus in **pregnancy** the os is soft and slightly patulous, whilst in **fibroma** the os is hard and firm, and, unless a submucous fibroid is descending, closed. In pregnancy there is the sensation of solid contents floating more or less freely in fluid, also contained within the uterine wall, and even in the early stage both os and body are soft. In fibroid

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degeneration the sensation is that of a solid, firm mass, solid throughout. The enlargement in both these cases is not tender, but it is markedly so in **acute metritis**, and even in chronic metritis there is usually more sensitiveness than is to be found in either pregnancy or fibroids. Fibromyoma, as a rule, causes very uneven enlargement of the uterus, producing large bosses. It only very exceptionally occurs that an evenly increased size is produced. When this is the case the two may be difficult to distinguish. Chronic metritis, however, whilst it produces some enlargement, never increases the bulk of the uterus to anything like the same extent as obtains in fibromyoma.

The uterus may be enlarged to some extent, but not very greatly, whilst remaining soft. It gives the examining finger the sensation of limpness and want of tone. There is usually associated with this a tendency for the upper part, the fundus and body, to bend over upon the lower, the cervix, in some direction, usually backwards. There is always a history of a previous confinement. It is **subinvolution.**

Projecting from the os may be a firm membrane containing fluid, the fœtal membranes in a threatened **abortion**; or in the same condition the membrane may have yielded, and its remains may be felt as soft and limp thin shreds hanging from the cervix. Hanging from the os may be a mass of rounded cyst-like bodies, resembling grapes, **hydatidiform mole**, or a firmer elastic body, which may be a **sarcomatous polypus**; or a still harder, firmer mass, a **fibromatous polypus.** A mucous or **glandular polypus** is extremely soft, and may possibly be felt only with great difficulty. The lips of the os itself may be studded with small hard masses, the size of a canary seed to that of a small shot; and the sensation to the examining finger is very much that of the presence of the latter. Associated with this the os is often felt to be split from below upwards, though not always. The little masses are occluded glands, distended by their own secretion, and the condition is known as **hypertrophy of Nabothian follicles.** They may be distinguished from the next condition by their minute size and very definite outline, as well as by their appearance on inspection.

More deeply seated and with less definite outline, blending indeed into the tissues around, a small mass the size of a pea or upwards may be felt, which will probably prove to be a commencing **carcinoma of the cervix.**

Projecting also from the tissues of the cervix, a large fungating, mushroom-shaped, soft mass may be felt—the **cauliflower form of carcinoma**; or the tissues of the os and cervix may be lost to some extent, so that the finger passes into a cup-shaped depression in one or other side, encountering firm, hard, rugged walls—the **ulcerative form of carcinoma.**

Before the finger reaches the os it may encounter at the orifice, or within the vagina, certain swellings. In one of the vulvar lips a firm elastic swelling may be felt. This may have a well-defined curved outline below, but no limiting line above. Pressure upon it will cause it to disappear upwards into the pelvis, with a gurgling sound. It is a **post-pudendal hernia.** The swelling may have no definite outline apart from that of the labium itself. It is doughy in consistence. It is probably **anasarca or hæmatocele.** The history and appearance will decide between these.

Or the swelling may be limited to the lower half of the labium. It has a well-defined outline all round. It is elastic and painless. It is a **cyst of Bartholini's gland.**

A similarly defined, limited mass, which has a more solid feeling, and is very painful on manipulation, is an **abscess** of the same gland. It is usually associated with gonorrhœa.

Passing up the vagina, the finger may touch a soft small mass at the urethral orifice, which is exquisitely tender. It is a **caruncle of the urethra.**

Still further up, and springing from one of the vaginal walls, to which it is connected by a pedicle, a rounded, soft, extremely elastic and thinly-walled body may be felt, occluding the lumen of the vaginal wall to some extent, but easily displaced out of the way. It is an **innocent cyst of the vaginal wall.**

The last nine conditions are scarcely within the scope of this paper, as they are detectable by touch with the internal hand alone, but they are added for the sake of completeness.

Returning to bimanual examination, if the internal finger is carried by the *side* of the cervix up to meet the external examining hand, it may find

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nothing resistant between them, in which case the broad ligament, tubes, and ovaries may be pronounced free from any growths, or their approximation may be prevented by a hard firm mass, the outlines of which are perfectly clear and separate from the vaginal wall. If this mass is equally firm throughout, and is bossed, it is almost certainly a **broad ligament fibroid.** If unequal in consistence, some portions being extremely hard whilst others are comparatively soft, it will possibly be a **dermoid cyst.** If the consistence is that of soft mud, and there are no definite separating outlines between it and the vaginal wall, which bulges somewhat into the vaginal canal, it may be a **broad ligament hæmatocele, or abscess.** Here, again, the history and other symptoms will decide between them. An abscess is always more tense and hot than a hæmatoma.

In all these cases, but especially in the fibroma and dermoid, the uterus will be pushed definitely over to the other side, or carried up out of reach of the internal finger.

The uterine outlines may be clearly definable, and the main portion of the broad ligament at the sides may be so free as not to prevent any approximation of the fingers, except at the upper extremity, where a firm transverse swelling may be felt. This is a **swollen Fallopian tube.** The swelling may be elastic and painless—**hydrosalpinx**; or firm and tender—**salpingitis**; in both of which cases the swelling will be more or less even from the uterus outwards. But occasionally a firm rounded swelling in its course may be felt more or less detached from the uterus, with still a cord-like connection with it; this is probably a **non-ruptured ectopic pregnancy.**

A **commencing ovarian cyst** may give almost exactly the same sensation to the examining hands, but it will not give the same cord-like connection with the uterus; it will be more elastic, and be painless. Ectopic pregnancy, on the other hand, is painful on pressure. The history and other symptoms are also different.

There are other rare conditions sometimes met with, but these are the most frequent, and to enumerate all would unduly prolong this lecture.

Of course I have not attempted to give all the symptoms associated with these tumours. My aim has rather been to show the kind and amount of information to be obtained by bimanual examination.

CLINICAL LECTURES
ON
DISEASES OF CHILDREN.

LECTURE XIII.

SOME FUNCTIONAL NERVOUS DISEASES OF CHILDHOOD.

Delivered at the London Hospital, December 6th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
Assistant Physician to the Hospital, and to the Hospital for Sick Children, Great Ormond Street, W.C.

GENTLEMEN,—In this and the succeeding lecture I propose to study with you some of the functional nervous disorders of childhood, which make up a group of diseases of great pathological interest and much practical importance. When you go into practice you will be struck by the frequency with which functional disease of the nervous system exhibits itself in your younger patients. One great reason for this no doubt is the comparative instability of the nervous system in early life. It is a nervous system which is, so to speak, still in the making. It has not yet settled down into the stereotyped form characteristic of mature life. The higher centres are as yet imperfectly developed, the lower incompletely controlled, and the paths of nervous impulses not clearly laid down. Hence abnormal forms of nervous action, manifesting themselves chiefly in defective co-ordination, spasmodic muscular contraction, and unchecked reflex excitability, readily become established. Several factors may come into play in rendering such abnormal action easier. First of these is a nervous heredity. Some babies are obviously "neurotic" from the moment of their birth. They are easily frightened, and start and tremble at any sudden noise or unaccustomed sight. Such children, you can readily believe, will easily become the subjects of functional disease of the nervous system. But more important, because more frequent than a neuropathic heredity, are two factors which by universal consent are admitted to play a large part in the production of the group of diseases at present under consideration.

These are—in young children *rickets*, and in older children *rheumatism*. Rickets and rheumatism are the predisposers *par excellence* to all forms of functional nervous disease. Why should rickets

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render the children who are the subjects of it more liable to suffer from nervous trouble? I think it is probably to be attributed to the fact that rickets is, after all, only a form of malnutrition; and all forms of malnutrition tend to affect the highest centres first; hence the cortex is weakened, and its control over the lower centres diminished. How rheumatism predisposes to these diseases I do not know, but one knows as a clinical fact that rheumatic children are much more subject to functional nervous disorders of all sorts than those who are not similarly affected. I would take chorea as an extreme example of that tendency. Chorea is only a functional disorder of the brain, of some part of the cerebral cortex, and it is almost invariably associated with rheumatism. Now chorea is a functional disorder which you are not likely to forget, because it is a dramatic disease and advertises its presence; but I want you to remember that what is true of chorea is true also of other less conspicuous nervous disturbances—namely, that they are specially prone to occur in children of rheumatic tendency.

Another point I want to insist upon before I proceed to deal with the different neuroses in detail, is the question of inco-ordination. When you study those nervous diseases more closely you will find that what you have to deal with in nearly every one of them is an inco-ordinate action of the nerve-centres. It need not be surprising to you that a lack of co-ordination forms the basis of most of these neuroses, because, as you know, a baby is to a large extent an inco-ordinate machine; at all events, it has not yet learnt to harmonise many of its movements properly. A young child has not yet learnt to co-ordinate and control the action of the expelling and sphincter muscles of the bladder and rectum; nor has it learnt to control properly the muscles which hold up the head. And not even by the time it is a year old has it acquired sufficient co-ordinating power to enable it to walk. So you need not be surprised that a mechanism which has been but recently and imperfectly acquired should be readily thrown out of gear, and that inco-ordination should be a striking feature of most forms of functional nervous disease in childhood.

I want now to pass to the study of some of those nervous disorders in detail. And first of all I wish to mention *facial irritability*, or Chvostek's sign. This is not a disease, but a symptom. It is a danger signal by which you recognise the tendency on the part of the child to suffer from more serious forms of nervous disorder. Facial irritability is so called because when you tap over the facial nerve, below the malar bone, you get a contraction of the facial muscles. The phenomenon is not peculiar to the face, for if you tap almost any exposed nerve you get a similar muscular contraction. In other words, there is an exaggerated excitability of the nerves to mechanical stimuli as the basis of Chvostek's sign. If that sign is present you know that the nervous system is in an irritable and unstable condition, and will be prone to be affected by convulsive and spasmodic disorders.

Let us take next *laryngismus stridulus*. That is a serious, and sometimes fatal form of nervous disease, which consists essentially in an inco-ordination of the muscles of the larynx. It is due to a spasmodic closure of the glottis, rendering the child for the time being incapable of breathing. Here you will almost invariably find that the child is suffering from rickets, that great predisposer to nerve disease. You will get the history that from time to time the child stops breathing, throws back the head, becomes blue in the face, and then suddenly emits a crowing sound, after which he may cry a little as if frightened, but soon returns to his toys. There is here, then, a period of apnœa—that is to say, cessation of breathing—followed by a crowing inspiration; and these are the characteristic signs of laryngismus stridulus. These attacks will come on more in the night and in the early morning than at any other part of the twenty-four hours; and they may easily be excited by any external irritation, by laughing, tickling of the throat, a draught of air blowing across the child's face when it is asleep, or by any emotional disturbance. At first the attacks occur only at rare intervals, but by-and-by they become more frequent, so that the child may have a great many seizures in the course of the day. What are you liable to mistake laryngismus for? First of all you might mistake it for *congenital laryngeal stridor*. I shall speak of that condition more fully soon. Meantime I will only say that congenital laryngeal stridor dates from birth, whereas laryngismus stridulus usually affects children who are about eighteen months old. So that proper attention to the history alone in these cases will keep you right in the matter of diagnosing between those two

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conditions. Next you will possibly mistake it for *croup*, the catarrhal laryngeal spasm of which I have already spoken; but croup attacks last for several hours, whereas laryngismus stridulus is almost momentary. Next you might conceivably mistake it for *whooping-cough*, pertussis; but whooping-cough is characterised by a series of short expirations followed by a whooping inspiration, whereas laryngismus stridulus is characterised by a period of apnœafollo wed by inspiration.

Laryngismus may be a serious disease, as I have said, and children sometimes die in an attack. Therefore do not be too confident in your prognosis. The story is told of a consultant who was summoned to see a case of this disease. He said there need be no alarm, as these children practically always got well; but as he was going out of the house he was called back to find that the child was already dead. We now come to treatment. First of all, how are you to treat an attack when it is in progress? What you have to do is to try to promote the taking of an inspiration; and this you can perhaps best do by dashing cold water on the child's face, or by tickling the fauces. If the spasm lasts an alarmingly long time, give a whiff of chloroform if you are able to do it yourself, or if there is some responsible person to whom you can entrust the administration. That will generally cause the spasm of the laryngeal muscle to pass off and re-establish the respiration. The application of a hot sponge over the larynx is also of use. Next, how are you to ward off future attacks? A great deal can be done by keeping the child quiet. It must not be annoyed in any way; it must not, as some one has said, be "tickled, tossed, or teased," because emotional disturbance and physical stimulation are exciting causes of attacks. It is well, also, to calm down the nervous system. The sedatives which you employ—and I shall have occasion to speak of them repeatedly—are bromides, chloral, and antipyrine. These three drugs are very useful in all the functional nervous disorders of childhood, and children stand all of them well, but particularly chloral, which you can always give boldly. Seeing that rickets is the great predisposer to those attacks, I need hardly tell you how important it is to treat the rickets. That is never to be neglected. It is absolutely necessary to recognise the rickety element and to treat it by a change of diet, by cod-liver oil, iron, and so on; and it is only in order to give those measures time to take effect that you meantime give bromides and chloral.

Next I wish to consider briefly the condition called *congenital laryngeal stridor*, which I told you might be mistaken for laryngismus stridulus. Congenital laryngeal stridor is a disease the pathology of which is still under dispute. It is believed by some to be due, like laryngismus stridulus, to an inco-ordination in the larynx. According to this view, it might be described as a stammer of respiration. When one makes an inspiratory effort, if the muscles are properly co-ordinated the vocal cords fly apart to allow the air to pass in. And those who hold this theory maintain that in congenital laryngeal spasm this does not happen. The child raises its chest, but does not at the same moment open its larynx, and it is only a second, or fraction of a second, later that the cords fly apart, and so there is a little catch as the air comes in. The other view—which is held by Dr. Lack, of this hospital, amongst others—is that you have to deal in congenital laryngeal stridor with a malformation of the larynx, consisting in a curving in of the epiglottis and flabbiness of the false vocal cords which allows the larynx to fall in upon itself during inspiration, and that it is in that way that the symptoms are produced. I shall not attempt to decide between these two views, but would say that it is admitted by all parties that the disease is congenital, and that towards the end of the first year of life it tends to disappear quite spontaneously. So far as I know it has never proved fatal. What are its symptoms? The child will be brought to you when quite a young infant, usually only a few weeks old, with the complaint that it makes a curious noise on breathing; and you will be able to hear a slight crowing or purring going on in its throat, similar to the noise which you may hear a cat make. This sound goes on more or less continuously, but is apt to be louder if the child gets excited or agitated. During sleep the noise is almost inaudible. Laryngismus stridulus, on the other hand, is not congenital, and it is essentially discontinuous. One other thing which you may mistake it for is adenoids in their congenital form, but I have already drawn your attention to that possibility. Inasmuch as congenital laryngeal stridor tends to disappear towards the end of the first year, it is not necessary to pursue any active

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treatment. I cannot satisfy myself that drugs have any effect upon it.

The next functional disorder of infants is the condition termed *tetany*, or *carpopedal spasm*, a name which describes the leading feature of the disease—namely, a spasmodic contraction of the flexor muscles in the front of the forearm and the back of the leg. You find the wrist flexed, and, as the interossei also participate in the spasm, there is flexion of the first phalanx and extension of the other two. In addition the thumb is often tucked in to the palm, so that the hand assumes a conical shape, which has been described as the "obstetric position," because that is the position in which the obstetrician puts his hand when inserting it into the vagina. You may also find a degree of œdema on the dorsum of the hand, and that the skin looks pink or purple and somewhat glazed, and that on handling it it seems to be tender. What this is due to I do not know. It may be produced mechanically by the continuous spasmodic contraction of the muscles interfering with the proper return of the blood. The position of the feet resembles that of the hands. The toes are flexed and curled into the soles, the heels drawn up, and the dorsum of the foot is often somewhat puffy. If you examine the electrical reaction of the affected muscles you find that their excitability is increased, and the anodal closing contraction is greater than the cathodal.

The great causes of this condition are two in number. In children of about eighteen months old it is very commonly associated with rickets. In younger infants you will find it coming on after an attack of diarrhœa. What the cause of the spasm is is not known. Sometimes you meet with a similar disease in grown-up people. It is one of the curious and rare complications of dilated stomach. It is assumed that in such a case you have to do with chronic poisoning, the result of the absorption of toxic material from the decomposing contents of the stomach. Arguing from that, some people say that tetany in young infants is produced in a similar way. It may be so, and its association with diarrhœa is suggestive; but there is no proof of it. Tetany is not a fatal disease, but it is a painful one. You must first of all treat the rickets, if there be any, and, whilst your rachitic treatment is taking effect, you must administer nerve-sedatives. And of those I would recommend the bromides and chloral in preference to antipyrine. Hot baths are also useful, for they tend to relax the spasm when it is very severe.

Lastly, I want to speak of a disease which is of considerable interest and rarity, and that is *head-nodding*. This condition of head-nodding has several synonyms. Sometimes it is called spasmus nutans, sometimes gyrospasm, and sometimes it is spoken of as simply rotatory head-spasm, or head-jerking, or head-rotation. Either head-nodding or spasmus nutans is the best term to use. In it you have to deal with continuous slight movements of the head, which tend to occur in young children who are usually about a year old, or a little older. When the child is brought to you the mother points out that the head is kept moving. Sometimes the movement is up and down, sometimes it is from side to side—a sort of pendulum movement,—and sometimes a combination of those forms in different degrees. The essential point is a slight more or less continuous and apparently involuntary movement of the head. When you come to examine these cases more carefully you will find that a majority of them suffer from nystagmus also. The nystagmus may be from side to side, or it may be vertical or of the rotary form, or it may be a combination of these varieties. And you may find in such a case a form of nystagmus which you do not find anywhere else,—that is to say, a form in which the eyes tend to jerk towards one another and then to fly apart. In most cases of nystagmus, as you probably know, the axes of the eyes remain parallel, but to this form the term "convergent nystagmus" is applied.

Here, again, it is believed you have to deal with an inco-ordination of the eye and head movements. The young child has just learnt to co-ordinate the movements of its eyes and head; it is just learning to turn its head in the direction in which it wants to look; and a very slight disorder may temporarily derange that co-ordinating mechanism. It has been compared to miner's nystagmus for a reason which I shall tell you of immediately. The interesting point which connects the movements of the eyes and those of the head is that the movements of the head tend specially to come on when the child tries to fix anything with its eyes. If you persuade the child to look steadfastly at you, you will find that the nystagmus increases and the head commences to nod. If the nystagmus is present

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in one eye only, the movements sometimes cease when that eye is covered up. This condition tends to occur with marked frequency at one part of the year—namely, in mid-winter. On a chart on the wall there are set out all the collected cases of head-nodding which are on record,* and you will observe that the maximum frequency is in the month of January. In the other months you will see there are hardly any cases at all. Why is head-nodding almost confined to this particular part of the year? An explanation which has been advanced is that it is due to the absence of sunshine at this time. And some of you who saw a recent case of the disease in the out-patient room will remember that the mother admitted that the child lived in a very dark room on the ground floor; and it is under such conditions that the disease most commonly appears. That is why people have compared this form of nystagmus with that known as miner's nystagmus, which is due to the constant strain of using the eyes in comparative darkness. Now, although that explanation is ingenious, and has much to commend it, it is not universally accepted, and in many cases it is impossible to find that the children who are the subjects of the condition have lived in dark houses; some of my cases have occurred in the suburbs or in the country, where there is as much light as can be met with in this country in the winter.

That brings us to speak of the diagnosis. The only condition for which you are likely to mistake head-nodding is *salaam convulsions*, which consist essentially of swaying or nodding movements of the body, which appear to be epileptic in their nature, one of the forms of *petit mal*. But head-nodding is associated with nystagmus, and occurs particularly at one time of the year, and tends always to pass off after a short time. There are certain automatic movements which idiots are apt to make which you may also mistake for head-nodding, such as swaying movements, banging of the head, or knocking of it against the pillow; but all these movements are much more gross, and are larger in their range, than the typical head-nodding.

The prognosis of this disease is perfectly good. If you leave it alone it will always get well in time. Therefore you can reassure the mother. Sometimes, however, the nystagmus seems to persist after the head-nodding has passed off; and it is possible that some cases of nystagmus which are alleged to have begun in childhood, and for which no cause is assigned, have been cases of nystagmus with head-nodding in which the eye movements have persisted.

The treatment which is most likely to cut short the movements and produce improvement is the administration of small doses of antipyrine—a grain for every year of life. This can be given two or three times in the day. I think I have seen this drug effect a distinct and decided improvement in the movements, rendering their occurrence less frequent and less marked; but the nystagmus seems to be more difficult to control.

* From a paper by Dr. John Thomson in Jacobi's 'Festschrift.'

In cases of senile pruritus, Jaeneche suggests regular brushing with a soft brush, which removes the degenerated superficial epithelial cells, increases the nutrition of the part, and brings about so much relief that the patients may go for days or even weeks with perfect comfort. The brushing is repeated at first three times daily for from ten to twenty minutes. These treatments are reduced to twice a day, and finally once every second day; a longer interval than this should not be allowed to elapse. There is often no improvement for days, nor is there any marked alteration in the nutrition of the skin. But shortly the patient experiences marked relief, and in many cases is entirely freed from suffering. It is important that no water should come in contact with the affected regions, since this, though it is followed by a temporary alleviation, renders the treatment by brushing quite futile.

The above is taken from 'The Medical Annual' for 1903, and admirably illustrates the object of this useful and practical publication by Wright and Company, of Bristol. The aim of the book is to present to the profession in a single volume annually a reflection of the knowledge of the year, and to condense and elucidate any new teaching that has come to the front at home or abroad. We have nothing but praise for the general summary of the year's work in the first part of the book, which concisely and clearly gives an idea of the general trend of medical opinion.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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LECTURES ON SOME AFFECTIONS OF THE HIP.

LECTURE I.

ON TUBERCULAR DISEASE OF THE HIP-JOINT.

By A. H. TUBBY, M.S., F.R.C.S.,

Surgeon to the Westminster Hospital; Senior Surgeon, Evelina Hospital for Sick Children, and Consulting Surgeon to the Hip Hospital, Sevenoaks.

GENTLEMEN,—I propose to devote myself at these demonstrations to the consideration of some affections about the hip-joint; and I will deal with a disease and afterwards with deformities. Of the diseases which affect the hip-joint, the most common is tubercular coxitis, and its very frequency is a sufficient excuse for me to say a good deal about it; and the lower one goes in the social scale the greater is the number of cases met with. It is a disease of young life, but not always so. I have seen it coming on after thirty years of age, and sometimes after forty, but then it is comparatively rare. As a rule it comes on some time before the fifteenth or the eighteenth year, and it appears with about equal frequency in boys and in girls up to the age of puberty; but after that age, as girls live a more sedate life and therefore are not so exposed to injury to their hips, the frequency is greater in boys.

With reference to the causation, I think one may say that though you hear so often of a history of injury given by the parents, traumatism is only an accessory cause. And if, after a slight injury, the hip-joint should show signs of loss of power and be painful, you may suspect very strongly that the child has, or has had, somewhere in its body the beginnings of tubercular disease, and that it would have developed tubercle in some place immediately a determining cause came into play; and the slight injury is the determining cause so far as the hip is concerned. It is difficult in all

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these cases to say if the hip-joint disease be primary or secondary. In some cases you will find, for instance, enlarged glands in the neck or some bone lesion co-existing, and subsequently hip disease comes on. Sometimes an enlargement of the psoas bursa precedes coxitis. But in a large number of cases the hip disease is the first sign of outbreak of tubercle. Unfortunately it is not by any means the last of the signs. I can recall a case in which a boy of seventeen had both hip-joints affected; they were ankylosed in a bad position, and then he developed very severe spinal caries. And it is almost impossible to determine, except where there is a previous lesion, whether the hip-joint disease is primary or secondary. The same difficulty exists about determining whether tuberculosis of the kidney is primary. It has been said that tubercular disease of the kidney may be primary, but it is very difficult to be certain on this point, because we know that some of these patients have had irritable bladders from tubercle, and then the infection spreads up the ureter, and so invades the pelvis of the kidney and infects it secondarily. With regard to the actual site of onset of hip disease, you may say that in children it usually commences in the bones. But about one sixth to one tenth of the total number of cases commence in the synovial membrane, and the remaining five sixths or more commence in the bones. Now as to the point of the commencement, we have certain epiphysial lines at the upper part of the femur. There is a well-marked growing line, the epiphysis of the head at its junction with the neck. There is a second well-marked epiphysial line, namely, the epiphysis of the great trochanter; and up to the age of eighteen there is a third epiphysis, the epiphysis of the lesser trochanter, which does not undergo ossification until nearly adult life. The most common place for the onset of tubercle is on either side of the epiphysial line of the neck. Another position where it begins is on either side of the trochanteric epiphysial line. And it may take different courses, depending upon its site of origin and upon its treatment. The cases in which the disease begins on the joint side of the epiphysis of the neck almost invariably result in infection of the joint. The disease goes on for some time on this side of the epiphysial line, and at last makes its way to the joint and opens into it, very frequently by a small aperture. I show a case here where it seems to me this has happened. Sometimes the disease is very extensive in its development at the epiphysial line, and then the whole of the head of the femur is cast off eventually as a large sequestrum. Such cases are comparatively rare, but are well known to occur, and when you do an operation for excision of the joint or open an abscess there, you may find the head of the femur lying loose as a large sequestrum; or another event will happen,—that is to say, the head will become fixed in the acetabulum, and separation of the neck will take place along the site of the disease. There are also some cases in which the disease begins on the shaft side of the epiphysial line of the neck. If the disease should progress towards the anterior surface of the neck, it always spreads to the joint, because the capsule of the joint on the anterior surface extends down to the intertrochanteric line. If the disease should make its way posteriorly, it need not necessarily enter into the joint at all, because the capsule does not extend so far outwards. And this is an important point to remember, because it is sometimes one's good fortune to be able to operate on these cases before the disease has perforated the joint. Then, again, there are cases beginning in the trochanter. They are always accompanied by symptoms very much like those of hip-joint disease, and they are extremely difficult to differentiate. But you may suspect such a thing as this is occurring, if the child at the beginning does not show very marked hip-joint disease, but if there is great thickening of the trochanter and much pain over it. Then you are probably dealing with a trochanteric epiphysitis, and there is ample justification for cutting down upon the trochanter and trying to find the focus, and clearing it out before disease makes its way into the joint.

From the trochanter or neck, infection may not only spread to the joint, but to the shaft of the femur. Fortunately it seldom happens now, but some years ago one saw many cases of amputation at the hip-joint for very severe disease of the femur; and you must always bear in mind the possibility that disease may not only spread to the joint, but that it may extend to the shaft, and so may give rise to that very terrible disease of the femur known as tubercular diaphysitis or tubercular disease of the shaft, which when once established is ineradicable except by amputation. That is an

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aspect of tubercular disease which I feel sure is not sufficiently appreciated, viz. the possibility of the whole bone being affected. And then it is not possible to do anything for it by surgery except to remove the whole of the diseased part.

Not only may hip disease begin in the femur, but it may also commence in the acetabulum. The cup of the acetabulum and the Y-shaped cartilages are liable to be the first seat of the disease. There is, as you know, a junction of three developing bones—the ilium, ischium, and pubes—at the bottom of the acetabulum, and it is very frequent to find disease beginning in the acetabulum. Some authorities say that it commences there as often as it does in the head of the femur, but in my opinion it is not so.

I do not propose to go through the whole list of *symptoms* this afternoon, but rather to emphasise certain points about them. Long before there are any local signs you will find constitutional signs, which you should carefully note. You will note that the child gets out of health, becomes pale, loses his appetite, and does not sleep well—a condition which is common to the onset of all tubercular troubles. But if you take the trouble to notice the child carefully and for a considerable time you will find every now and then that the child limps in a characteristic way; for instance, he will limp towards the end of the day, and not so much at the beginning. These facts alone ought to point to you the possibility of tubercular trouble being present there. Then you have the *local signs*, which I think every one here is acquainted with. But the striking point about hip-joint disease is that there is no one sign which is absolutely diagnostic of it; it is rather the grouping of the signs, if I may say so, which enables us to make a correct diagnosis in these cases. First of all there is the question of pain, and the pain varies very much in hip-joint disease. We were taught in our anatomy classes that it was very frequently felt at the knee-joint, but this point has been much exaggerated; pain is not present in the knee-joint in anything like the number of cases you would be led to suppose from reading books. As a rule the pain is more marked in the neighbourhood of the hip-joint, and it may be felt in varying situations. If the disease is making its way anteriorly you will find there is some pain in the neighbourhood of the femoral artery. If, on the other hand, it is commencing in the trochanter, you will find there is pain on pressing gently over the trochanter. If the disease be acetabular or is making its way posteriorly you will note pain in the neighbourhood of the sciatic notch. So that the position of the pain may give you some idea where the disease is going on. The pain is, as a rule, worse when the child is moving about, excluding that which is associated with starting at night, and with "night-cries." The explanation of them I believe to be the following:—If the disease makes its way towards the joint cavities, after a time it causes a certain amount of inflammation and erosion of the bone surfaces. Then the cartilage is raised, and so a tender spot is formed. While the child is awake all the muscles are at attention, so as to prevent intra-articular pressure. But when the child falls asleep the joint surfaces are brought into contact, and hence the pain. With reference to the pain in the knee, this often leads to mistakes in diagnosis. Here is a case in point which I have brought for you to see. This child has been ill one year, and her trouble began with starting pains in the knee. She was taken to a certain hospital, and in the out-patient department a splint and Scott's dressing were applied to the knee. She had been attending the out-patient department of another hospital since September, where, after a week or two, she was taken in and put to bed, and the pain in the knee and hip disappeared. Contrary to the usual rule, this was a case in which the pain in the knee was more striking than the pain in the hip-joint; so much was this so that the case was treated as one of knee and not of hip trouble. But, considering how insistent the pain in the knee was in this case, one hardly wonders that the mistake arose.

Now we come to the special deformities of coxitis. Their explanation is difficult. We were taught that in the early stages the deformity of the limb was one of abduction, rotation outwards, and apparent lengthening.

That is nearly correct; and in the second stage you have the reverse condition—adduction, inversion, and apparent shortening. What is the explanation of the conditions of abduction and apparent lengthening? It was supposed that the capsule of the joint at that time was full of fluid, and it was shown experimentally that if you take a pelvis with the femur attached, and the liga-

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ments and capsule of the joint intact, and trephine over the site of the acetabulum and fill up the joint with fluid, the limb becomes abducted and everted. But, unfortunately, this explanation will not do for all the conditions which we meet with in hip disease, because in the second stage it is very certain that the joint is also full of fluid, and yet the limb in this case is adducted and inverted; and therefore the fluid theory does not explain why in the first stage there are abduction and apparent lengthening, and in the second stage adduction and apparent shortening. I take it, however, that the explanation is this: as to whether you get adduction or abduction depends very much on whether the child goes about with crutches or without. If the child suffers from hip-joint disease and he should walk without crutches, in order to spare the joint as much as possible he will flex the knee and bring the foot out as much as possible, and raise the heel so as to give it a firm but elastic base for the moment when he bears its weight on the affected side as he limps along. This explains the abduction, and with the abduction there must necessarily follow the apparent lengthening. So that abduction arises firstly from the tiptoe tendency, and secondly from the necessity for a wider base of support in walking. But when these patients come to walk with crutches the conditions are very much altered. They partially bear the weight on the diseased side by means of a crutch on that side, and they incline the pelvis well towards the sound side, so as to bear the weight as much as possible on that side and take it off the diseased. Then the affected leg is free to drop inwards; hence the adduction. This is, I think, a very fair explanation of why you get abduction in one stage and adduction in the other. It depends whether the child is progressing with or without crutches, and you may see cases from the first, some with adduction and some with abduction.

With regard to the actual signs about the hip-joint, there are two which should always place you on your guard. They are difficult to elicit satisfactorily, but at the same time there is a proper way of ascertaining whether they are present or not. In some cases you will notice as soon as you see the patient that there are flexion and lordosis. In other cases these are comparatively slight, and in order to find out whether there is rigidity about the hip-joint you have to proceed gently or you will cause considerable pain. The proper way to examine for flexion and rigidity is not to take hold of the presumably affected limb and attempt to raise it up, but to take the sound limb, firstly to reassure the child, and secondly to judge by flexing up the sound limb what the amount of flexion deformity is on the affected side. For instance, suppose the right to be the affected side, and you wish to ascertain if there is flexion, and what is its amount. Let the child lie supine and then put your hand beneath the back and flex up the left side until the back touches the bed, and if there be spasm present the right side begins to flex up more and more. So by taking hold of the sound side in that way and flexing the limb you ascertain the degree of flexion in the affected side, and you do it without causing pain. There is a little flexion in this case, but even before definite flexion is present you will find there is some rigidity. Of all the early local signs of hip-joint trouble, there is no doubt rigidity is the first, and sometimes it is very difficult to elicit it satisfactorily. But if you have a case in which you suspect there is joint trouble, when you take the limb and bend it up very slowly, not jerkily, you will find frequently that you cannot get a full degree of flexion. Of all the valuable signs of diagnosing hip-joint disease the loss of complete flexion is perhaps the most so, and with the loss of flexion there is also rigidity. Another sign which is very much relied upon is loss of rotation, and it is said by some—and I am inclined to support the belief—that if you can place both limbs in the flexed and fully abducted and externally rotated position, there is no hip-joint disease present.

Having ascertained, say, in a given case, if there is flexion deformity, with abduction or adduction, as the case may be, and hearing that there is pain, you now proceed to ascertain whether there are any of the consequences of hip-joint disease present—namely, abscess or fixed deformity. I believe one case in six to one case in ten of tubercular coxitis is followed by abscess, and when an abscess appears, it necessarily follows that the prognosis is very much worse, and for this reason. Abscess elsewhere is not a matter of so much importance, but abscess in the neighbourhood of the hip-joint is very liable to be contaminated

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with septic material; and if it once becomes so I think it is almost impossible to get it thoroughly clean again. For that reason alone, therefore, apart from the necessary drain which the formation and discharge of an abscess must be, it adds enormously to the risk of hip-joint disease. At one time it was almost a fatal complication. Now it is not so, because we have learnt to treat abscesses on a different plan, and there is not so much drainage-tubing used. With regard to the situation of the abscess, this depends to a certain extent on the site where the disease has broken down, and also upon the way in which pus tracks through the muscles. Thus there may be an antero-internal abscess, internal to the femoral artery; or an antero-external abscess, external to the artery, or an abscess over the trochanter; or there may be a pelvic abscess coming out through the sciatic notch, or appearing above Poupart's ligament. The latter is said—and I believe it is true—to indicate disease of the acetabulum, and probably perforation of it. In order to ascertain the extent of pelvic abscesses it is absolutely incumbent on you to examine *per rectum*. By this means you can ascertain their extent and size, and very often their point of origin. This form of abscess is formidable partly because it spreads very extensively, and partly because it opens in the neighbourhood of septic centres. Then it is very difficult to drain, and I am inclined to regard cases in which there is definite disease of the acetabulum as more serious than when the disease begins in the upper end of the femur.

A deformity from hip-joint disease may be either temporary or it may be permanent. It is very common for children who have been going about with a painful joint to be sent into the hospital with a certain amount of flexion and a certain amount of spasm, all of which will disappear, as you see in this case. This child was admitted because of flexion and spasm, and if you look at her you will find the position of the legs is now normal after appropriate treatment.

I will now pass from temporary deformity and speak more particularly about pathological dislocation. As a result of the inflammatory process all the ligaments become softened. Sometimes suddenly, but more often gradually, the head of the femur leaves the acetabular notch; and I have seen two, if not three cases, perhaps more, of sudden dislocation of the head of the femur arising in connection with hip-joint disease. In fact, dislocation may occur in a single night, and I believe it occurs in the night-starts, or it may be that what is left of the head slips away from the acetabulum, and you find it on the dorsum ilii, simulating a traumatic dorsal dislocation. More often, however, the process is gradual, and there is a very interesting specimen from a case of the late Professor Volkmann, of Halle, which shows the successive positions occupied by the displaced head. The acetabulum was very much widened, and there were successive positions where the head had been, and this interesting condition he called a "wandering" acetabulum, which is extremely expressive. It gives you the right idea, although it should be termed a wandering head of femur, and it further shows the widening of the acetabulum and the formation of more or less imperfect facets where the head of the bone had been lodged. Ankylosis, fibrous or bony, takes place after dislocation, and gives rise to permanent deformity of the hip-joint. So hip deformities may be either temporary or permanent.

The *diagnosis* of coxitis is often a very difficult matter. Unfortunately it is the most common affection in this neighbourhood, and it is one of the most serious. There are three main conditions from which you should diagnose hip-joint disease. These are congenital dislocation of the hip, paralytic dislocation of the hip, and coxa vara. With reference to congenital dislocation of the hip, there are certain points which are common to it and to ordinary tubercular disease of the joint; in both you may find the head of the femur out of place, in both you will see a certain amount of flexion, and in both a certain amount of eversion, as well as some shortening of the limb. In congenital dislocation the movements are unaccompanied by any pain whatever, and, as a rule, there is no spasm of muscles; but in cases of old standing there is often contraction of the adductor and flexor muscles of the thigh. But the most striking point about congenital dislocation of the hip is that you can by pulling on the leg alter the level of the head of the femur, which you can never do except under an anæsthetic in hip-joint disease. So the striking point in diagnosis is that in congenital dislocation of the hip you have freedom from pain and power to "telescope" the femur up and down.

With reference to coxa vara, the diagnosis in

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early conditions is a difficult matter, and I shall have to speak of it more fully, because I propose to devote the greater part of my next lecture to the subject of coxa vara, it being a newly recognised and somewhat difficult condition to diagnose.

Then there is paralytic dislocation of the hip. I have seen three cases of this sort,—that is to say, sudden dislocation of hip arising in connection with infantile paralysis. The particular case of which I show the skiagram, I think, was one which, after failure in manipulation, I reduced comparatively easily by making an incision down to the trochanter, carefully separating the muscles, and dividing the tendon of the obturator internus. The head went in without difficulty. Hip-joint disease in children has been mistaken for appendicitis on the right side; and *vice versâ*, pelvic abscess from coxitis has been mistaken for appendicitis. Pelvic abscess is difficult to diagnose from appendicitis unless you take the proper precaution of going into the history and examining *per rectum*. Sacro-iliac disease in its early stages may give rise to difficulties, but it can be excluded by carefully noting the history, the attitude, the effect of lateral pressure on the pelvis, and the fulness or otherwise over the joint.

There are several other conditions which are mistaken for hip-joint disease, and some of them are very difficult to diagnose. Suppose you have, as in this next case, osteitis in the great trochanter. In such an event there will be all the signs of hip-joint disease, and it is almost impossible, in tubercular osteitis of that part, to diagnose it for a certainty from ordinary hip-joint disease. But remembering what I said just now, if in a case of supposed hip-joint disease there is rapid and early enlargement over the great trochanter, with much pain, it behoves one to operate as soon as possible and get rid of the disease in the trochanter so as to prevent it spreading into the joint. I believe many joints have been lost through want of early operation in these particular cases.

Another case I had, which to my mind illustrates the necessity of early operation—not that I advocate it in all cases,—was of a child in whom there were all the symptoms of hip-joint disease. I cut down on the joint and found an aperture in the bone external to the capsule which communicated with a large cavity in the head. I carefully scraped out the cavity and stuffed it with gauze soaked in menthol solution, and then, having disinfected my finger, I opened the joint and found it quite free from disease. I closed the opening in the capsule with a few silkworm gut sutures, and that child has done well and recovered with a very movable hip-joint. In fact, the disease was taken in time. *What we should aim at in hip-joint disease is the early recognition of extra-articular foci and their treatment as soon as possible, so as to prevent disease spreading to the joint.* There are many other conditions which may cause trouble, but it is wearisome to go into a number of possibilities which are all set out in the text-books.

Now with reference to *treatment*, we may take first of all the treatment of uncomplicated cases. I am constantly asked, in consultation and by students, how long will the child have to lie up? The more experience one has of tubercular affections, the more cautious and wary one becomes in giving prognosis as to time. I used to say an inflamed hip should be treated for two years, but I am now convinced that to completely cure a hip-joint case you must keep it for three years under observation. I do not say the patient should be lying down for all that time, but I become more and more cautious, because I find cases which do not follow expectations. The patient is allowed to get up too early and he soon relapses, and then you find you will have to begin the process of treatment over again, with the result that the cure is delayed. So if you have an uncomplicated case of hip-joint disease to deal with in a weakly child, you may say that that case will require three years of treatment, and possibly longer, even in the event of no abscess forming. There is a great tendency on the part of these tubercular cases to relapse, and if they do so they are much worse than when the treatment is carried on uninterruptedly from start to finish. I think one should insist very strongly that these cases cannot be cured by three or six months' rest in bed; they are apparently cured for a time, and they begin to go about as ordinary people, but then they relapse, and their last stage is worse than their first. Time is lost ultimately by this kind of treatment, because there has been too much haste to cure the case. It requires a good deal of moral courage to insist upon treatment extending over several years, but it is the duty of the surgeon to do so.

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We will now take an uncomplicated case in which the child is brought with a certain amount of lordosis and flexion. I shall speak decidedly on this point once for all. If there are pain, flexion, lordosis, and wasting in a patient, it is futile to treat that child by the ambulatory method, —that is to say, to allow it to walk about. It is a common practice to put on a Thomas's splint, and a large proportion of them do not fit at all, and therefore are useless. The child walks with crutches, goes about in pain, spoils the hip still further, and he is worse at the end of this ineffectual treatment than at the beginning. I think all cases of this kind should be put to rest at once; and, as an adjunct to rest, a very important thing is extension. There is a point which was advanced by Mr. Howard Marsh at the Alexandra Hip Hospital many years ago, and I used to see it very largely in practice when by his kindness I attended there. I do not know how many times I have seen—and you must also have seen—a child put to bed with a weight fixed to the leg irrespective of the position of the limb. It is true the flexed limb is brought down in the bed, but the lordosis and obliquity of the pelvis remain unreduced. The result is the child has night-cries and continues to waste. I take it that the result of weight extension applied in this way is to increase the spasm of the psoas and flexor muscles and cause the bones to be pressed firmly together at the site of disease. The proper way to apply weight extension is as follows:—An adjustable inclined plane should be made. It must not be too wide, but just sufficiently so to accommodate the leg with the knee fully extended comfortably, and it should have attached to the distal end a pulley to carry the extension cord and capable of being moved laterally. After applying the stirrup to the affected limb, its true position is ascertained thus:—It is brought inwards or outwards until the anterior superior spines are level, and it is then flexed up until all lordosis has disappeared and the lumbar spine is flat on the bed. If you take care, before applying the extension, to flex the thigh up a little more than is sufficient to entirely overcome the lordosis, and so relax the psoas, you will find the muscular spasm ceases in a few days, night-cries no longer trouble the patient, and the child begins to pick up and to put on flesh. This means that the irritation at the diseased joint is subsiding and the spasm is disappearing. After a few days you may lessen the angle of the inclined plane without any disturbance, and in ten to twenty-eight days it will happen that all the flexion and adduction or abduction deformity have disappeared and the limb lies parallel with its fellow; and it can be retained thus by weight extension without pain, and the joint is allowed to settle down in the best position possible. Very large angles of flexion may be reduced in this way, and the child I show you now, with quite a straight limb, came into the Evelina Hospital with the hip flexed at more than 45°. But I have never seen extension succeed when it has been put on in the faulty way so often practised. A word as to the number of pounds weight which should be applied. For a child of three years two and a half pounds are sufficient, and a good rule is to increase the weight by half a pound for every year of age beyond this, up to five or six pounds. But in cases even of children of seven to ten years, always begin with a small amount of weight at first. Some surgeons prefer to extend the hip under an anæsthetic at one setting, but I cannot help thinking this is adding risk to the case.

As to the treatment of abscess, I wish to say this, and it is important:—It should consist in opening the abscess, cleansing the abscess cavity thoroughly, and, what is essential, *sewing it up at once*. I know that is very unusual, but the great danger to be feared in these cases is that there may follow streptococcic and staphylococcic infection superadded to the tubercular infection. The way I always treat my cases of hip-joint abscess, and, as far as possible, those of spinal disease, is as follows:—After careful sterilisation I open them pretty widely, and I have no hesitation in making two or three openings at some distance from each other for better access to the cavity, and then I scrape out the whole of the sac and get out everything I can. Then it is well flushed out with sterilised water, and thoroughly dried with sponges on sticks. After that I swab out the cavity with a solution of menthol and glycerine in the following proportion:—Menthol, one drachm; spirit one ounce; glycerine, to one pint. This preparation seems to act extremely well in these tubercular cases. Then I express all the superfluous menthol solution and close the wounds up, and

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afterwards apply firm pads over the site of the abscess cavity, and immobilise the part. I do not expect that there will always be primary union, for at one spot where an opening has been made there is sometimes a little gaping and the appearance of a small amount of fluid. On putting a sterilised probe in, there issues not pus but serous fluid, which I believe is due to the irritation caused by the solution, and possibly some of the non-absorbed menthol. After that the place quietens down and heals by primary union. If it does not, you can open it up again, and you are much better off even then than if you allowed it to be open from the first, for the patient is not running the risk of septic infection, the chief thing to be guarded against and avoided.

With regard to the treatment of severe deformity of the hip-joint, when the joint has once got ankylosed, I do an Adams's operation in preference to Gant's infra-trochanteric osteotomy, and I now show to you a patient on whom I performed Adams's operation. She came under my notice some time last year, and she had much difficulty in walking. She was bent down a good deal, and there was flexion of the hip almost to a right angle. So in her case I performed an Adams's operation, which consists in sawing through the neck of the femur and putting the shaft in a right line with the pelvis. Thus you obtain ankylosis in a straight line and not at a right angle. You must never expect to get a false joint to form, because experience shows that it is impossible. Although there is some difference in the length of the legs of this patient, she can walk well.

A few words with regard to excision of the hip. As a formal operation, I am opposed to it. Some results are good, but the majority are not satisfactory, because at the operation, in sawing through the femur, new and probably healthy bone is exposed and inoculated, and the after results, so far as locomotion and power at the hip-joint are concerned, are far from satisfactory. There is only one justification for a formal excision, viz. when it is desired to obtain ample room to clean the acetabulum. But the better operation is erasion of all the diseased parts, and when the disease in the femur has been scraped away there will be sufficient room to reach the acetabulum.

Formerly I have seen amputation performed fairly frequently for extensive disease of the pelvis, and tubercular diaphysitis of the femur, sinuses, and oncoming lardaceous disease. But fortunately the cases in which so drastic a remedy is called for have diminished, greatly owing to improved methods of treatment.

Treatment of Furunculosis.—Cohn suffered from a remarkably severe attack of furunculosis himself, and perseveringly applied all the ordinary remedies without avail. Even yeast and a four weeks' course at the strongest sulphur baths in Europe (Nenndorf) proved ineffectual. He attributed the primary infection to a flea-bite, either from germs transmitted by the insect or introduced in scratching. The following method of treatment finally proved effectual on himself and has since been regularly employed in his practice, with results far surpassing those obtained by the other methods in vogue. As soon as the incipient pimple or furuncle shows itself he anoints it with a little ichthargan salve, from the size of a pinhead to that of a bean, rubbing it first into the vicinity and then over the lesion itself two or three times a day. All internal medication is avoided, except that the bowels should be regulated and, possibly, sulphur waters or arsenic be taken. The formula for the salve is 10 grms. ichthargan, 5 grms. aq. dest., 10 grms. glycerine, 35 grms. lanoline, and 40 grms. vaseline. When the inflammation has subsided, a 1 per cent. ichthyol paste can be substituted. Furuncles on or near the genitalia require caution, as the ichthargan is not able to display its full power, and the ordinary measures may be preferable. Cohn warns against the use of the knife except in case of a large carbuncle. It is then too late for ichthargan treatment, and he uses a platinum cautery and applies a salicylated soap plaster and zinc plaster covering. As an adjuvant he recommends a daily bath, with 60 grms. ichthyol in the water or sulphur baths. If the expense is prohibitory, he advises Arning's creolin baths. A course at Nenndorf, continued, if necessary, for six weeks, may possibly be useful.

Sodium Cacodylate in Dermatoses.—Pabis found this drug effective in improving the general nutrition in cases of eczema, ichthyosis, prurigo, and lupus, and thus indirectly benefiting the dermatosis.—*Journ. A. M. A.*, May 9th.

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CLINICAL LECTURES
ON
DISEASES OF CHILDREN.

LECTURE XIV.

SOME FUNCTIONAL NERVOUS DISEASES OF CHILDHOOD—*continued.*

Delivered at the London Hospital, December 9th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
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GENTLEMEN,—In the last lecture we began the consideration of a group of functional nervous disorders in children, the essential nature of which, we saw, was that they consist in an inco-ordination of movement. We saw, further, that there were several reasons why children are particularly subject to such inco-ordinate action of muscles, one of the chief being that, in the case of young children at any rate, co-ordination is a recently acquired function of the nervous system, and therefore is one which is very easily lost under adverse conditions. We saw that amongst such adverse conditions must be placed many diseases of nutrition, rickets being one of the most conspicuous. In addition it was pointed out that in older children rheumatism plays a part in the production of functional nervous disease, and that the factor of heredity can never be altogether ignored. We reviewed some examples of functional nervous diseases, taking up first the diseases of the larynx—congenital laryngeal stridor and laryngismus stridulus,—and then we passed on to tetany and head-nodding. I wish to-day to conclude this subject with you, beginning with the important question of convulsions.

You will meet with *convulsions* in children very frequently in your practice, and they are very alarming to the parents of the child. And further, from the point of view of the medical man, their exact causation is often difficult to diagnose. I want first of all to warn you that mothers and nurses will call by the name of convulsions many things which really have no right to be so called. They will tell you, for instance, that the child is suffering from "inward convulsions," or "inward fits," or "screaming convulsions." And they may mislead you into thinking that you have to do with a case of genuine convulsive disease. Many children under the irritation of teething or colic, or under emotional disturbance, exhibit twitchings of muscles, particularly muscles of the face, which the parents are apt to suppose are true convulsions. But you are not to regard mere quiverings of eyelids and screwing up of the mouth, or violent fits of screaming, as genuinely convulsive, though they may be of value to you as warnings that a tendency to convulsions exists.

You will find it convenient to divide genuine convulsions into two main groups—first, those which are *idiopathic;* and secondly, those which are *symptomatic.* By idiopathic convulsions one means those for which one can find no cause, which arise in a child who is apparently in fairly good health, and which may pass off after a time, or which not infrequently become established, so that the child suffers from fits from that time onwards at varying intervals. There can be no doubt that a considerable number of those cases are examples of genuine epilepsy. Epilepsy may certainly begin in early life, so that when you are called to a child which is suffering from violent convulsions for which you can find no cause, you must bear in mind that you may be dealing with the first stage of that disease. Sir William Gowers puts the proportion of cases of epilepsy beginning as convulsions in infancy at 10 per cent., and some authors give an even higher figure.

Far more important, because more common, are cases of the symptomatic group. In the first place, you may have symptomatic convulsions as the equivalent of a *rigor* in a grown-up person. Many young children, instead of having a rigor at the outset of an acute illness, have actually a general convulsion. This may happen at the commencement of pneumonia, or scarlet fever, or any of the other acute specific fevers, where the temperature runs up rapidly during the first few hours of the illness. Secondly, symptomatic convulsions may be an indication of *organic disease of the brain.* They may occur in meningitis in all its forms, or they may be met with in cases of cerebral tumour, or with thrombosis of cerebral veins. Indeed, in almost any serious intra-cranial disease in children you may find convulsions as one of the symptoms. Thirdly, convulsions may be of *reflex* character or origin, an expression of the reflex irritation which arises from such things as the cutting of teeth or the existence of colic or phimosis. The reflex

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irritation which results from these causes may, as it were, "fire off" convulsions from the cerebral cortex. There is another group which is *toxic* in origin. Uræmic convulsions are a typical example of this group; and I think it is not unlikely that some of the cases of convulsions which you meet with in the course of acute diarrhœa can also be attributed to toxic causes. Lastly, there are the convulsions which are the result of *asphyxia*. Such are those which you may sometimes meet with in the course of pertussis. Towards the end of any illness, also, convulsions may come on and be, so to speak, one of the modes of dying. These also are probably due to failure of respiration or of the heart, leading to cyanosis and congestion of the brain.

Those are the chief groups into which you can divide cases of convulsions; and when you are called to a case you have to decide as far as you can to which one of these classes it belongs.

You will find that, just as in many of the other functional neuroses we spoke of last time, so here rickets is the great predisposing factor. For instance, the cutting of teeth in a child in good health may not set up convulsions, but if the child be rickety, such irritation may be a determining cause of spasmodic discharges from the cortex of the badly nourished brain. The same is true of the other convulsions of which I have spoken. Many of them are insufficient by themselves to produce convulsions in an ordinary child, but where rickets has produced nervous instability they may suffice as determining causes. You can sometimes perceive that convulsions are going to come on by certain warning signs. One of these is the development of the facial irritability of which we spoke last time, and which we saw to be an indication of undue irritability of the nervous system. Another will be slight twitching movements of the face and extremities. If these twitchings are seen in a child the subject of rickets you ought to anticipate that the child may become affected by convulsions, and do what you can to ward them off. Sudden flushings or attacks of coldness are also occasional premonitory signs.

With regard to *treatment*, your immediate line of action when called to a child who is at that moment the subject of a convulsion must be the same no matter what the cause of the attack may be. First of all you lay the child down, raising the head somewhat, and loosen the clothing just as you do in a case of epilepsy, so that there is no hampering of respiration. And it is a good thing, unless you have been anticipated by the mother or nurse, to put the child into a warm bath, though this is perhaps most efficacious in those cases which are the result of colic. Next you should give sedatives by the mouth, or, if you are unable to get the child to swallow, by the rectum. Chloral is the most efficient, three or five grains by the rectum to a child six months old. If you fail, and the convulsions are very serious and still persist, and the friends are getting alarmed, there is one resource which is open to you, and which I think is practically certain to be successful—namely, the inhalation of chloroform. A few whiffs of chloroform given on a handkerchief will cause the convulsions to cease by paralysing the brain cortex. Such will be your immediate treatment in all cases. Next you have to consider how you are to prevent the recurrence of the attacks. In order to do that you must treat the rickets, if there be any. You must make a radical alteration in the child's diet and administer cod-liver oil. But occasionally, and until these measures have time to take effect, you may have to diminish the excitability of the nerve-centres by sedatives. Bromides and chloral will be the drugs which you will use. I would remind you again here how well young children stand chloral; it is a drug which you may give pretty freely without risk. And in those cases it is to be preferred to bromide, as being more efficient in controlling the convulsive tendency. It may be necessary, of course, to attend to the child's digestion if there be colic, and that may involve the alteration of the diet in directions I have laid down in a previous lecture.

When you consider the *prognosis* of convulsions you have to take into account what the immediate chances are, and also what is likely to be the remote outcome of the seizure. Children do not often die in a convulsion, unless the convulsions are themselves merely the final scene in an already mortal complaint,—that is to say, unless they are simply a symptom of the terminal asphyxia. Convulsions as a disease are not immediately fatal. You have to remember, however, that in a considerable proportion of the cases the convulsions may persist in the form of epilepsy; and you have further to remember that nearly all, or quite a large number,

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of the children who suffer from convulsions manifest in later life nervous disorders of different sorts. Of about eighty-five cases followed up by Dr. Coutts and traced into adult life it was found that eleven suffered from major epilepsy, four from petit mal, five exhibited somnambulism, four developed melancholia, seven had chorea, and nine suffered from migraine. So you see in what a large proportion of those cases the later history of the patient is marked by further nervous disturbance.

I now pass to another extremely important group of nervous disorders, those which go by the name of *night terrors*, or *pavor nocturnus*. Night terrors of varying severity occur with a considerable degree of frequency in children, and they are often extremely alarming to the friends. The usual history of a case is that a child, most commonly about the period of the second dentition, falls to sleep and shortly afterwards wakes up in a violent state of terror; he sits up in bed and shouts in an incoherent manner, perhaps only partially awake, and it may be a long time before the mother or the nurse succeeds in pacifying him. In those cases the cause of the fright is sometimes an hallucination of vision; sometimes, on the other hand, it is probably merely a nightmare. It has been said, I think by an American writer, that you may divide the cases into two groups—"those that see visions, and those that dream dreams." Those who see visions are those who have hallucinations of sight; those who dream dreams are those who have what is equivalent to a nightmare in the adult. Although that is probably a real distinction, you will find it extremely difficult to say to which of those two classes any given case belongs, because these children are often incapable, sometimes unwilling, to give an accurate description of their sensations during the attack. Sometimes they will tell you that they see frightening objects, shadows or dark shapes in the room, and in such cases perhaps they are the subjects of visual hallucinations. In some cases they can give you no coherent account of their sensations. These experiences may be repeated night after night, until the equilibrium of the nervous system is seriously upset, and the child begins to dread going to bed at all; and then the friends get alarmed and think that the child is becoming insane, and call in a doctor.

A very good description of the sensations of children who suffer from those attacks has been given by Charles Lamb in one of the 'Essays of Elia' entitled "Witches and other Night Fears." Perhaps the passage is known to some of you. He says: "I was dreadfully alive to nervous terrors. The night-time, solitude, and the dark were my Hell. The sufferings I endured in this nature would justify the expression. I never laid my head on my pillow, I suppose, from the fourth to the seventh or eighth year of my life [*i. e.* about the time of his second dentition], so far as memory serves in things so long ago, without an assurance, which realised its own prophecy, of seeing some frightful spectre. . . . Parents do not know what they do when they leave tender babes alone to go to sleep in the dark. The feeling about for a friendly arm, the hoping for a familiar voice when they wake screaming and find none to soothe them—what a terrible shaking it is to their poor nerves! The keeping them up till midnight through candle-light, and the unwholesome hours, as they are called, would, I am satisfied, in a medical point of view, prove the better caution."

Now, gentlemen, night terrors may be taken as always evidence of ill-health in some form or other. They do not occur in perfectly sound children. And you will find that amongst the predisposing causes particularly at work are, in the first place, a neurotic or nervous heredity; secondly, you will find a rheumatic diathesis, as I have already stated. But above all, and far more important than all, perhaps, is over-pressure at school. One of the chief predisposing causes of night terrors is school work, and you will often in many cases find that children only suffer from this disorder during the school term. During the holidays they sleep undisturbed, but when they resume school they resume also their night terrors. Mothers often describe them as "going over their sums in their heads all night," arithmetic being apparently one of the chief things which exhaust and irritate the brain.

Of exciting causes I would emphasize two which it is of the very first importance for you to be familiar with. One of them is gastro-intestinal disorder, especially *constipation*. The second is partial asphyxia from the presence of *adenoids* in the throat. These two causes are by far the most important for you to recognise from a practical point of view.

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When I have mentioned to you the predisposing causes of night terrors, you see what the treatment should be. It must not be purely sedative. There are two things above all that you should do when called to a child who suffers from this complaint; one is to attend to the state of the throat, and the other is to administer aperients. If you do these you may leave almost everything else undone. Clear out adenoids when you find them, and give an aperient, no matter what the state of the bowels may be or is said to be. If you do that you will not go far wrong in the management of those cases. There are other accessory means which are of help. These children ought not to be frightened in any way, nor excited. They ought not to be allowed to sleep alone, they ought to have a night-light in the room, and ought not to be allowed to go to bed with a full stomach, because, as in night-mare in grown-up persons, so here, pressure of food in the stomach may be a cause of the condition. It may be well to remove the child from school for the time being, until the nervous system has recovered its equilibrium. It may be necessary to give sedatives, such as bromides and chloral, temporarily; but as far as possible you should dispense with these and direct your attention to putting the child into that state of health in which night terrors are impossible; and in order to do that, as I have said, you must pay attention to the state of the throat and stomach and bowels.

There is an analogous condition to night terrors which occurs in the day, called day terrors, or *pavor diurnus*, which is very much rarer. The child, in the middle of its games, may be seized with a fit of panic and rush to its mother, giving vent to inarticulate cries. The fit of terror may last for a few moments. These are no doubt to some extent analogous to the worst form of night terrors. They are probably cases where there has been some hallucination of some of the sense organs, and must be treated on the same lines as the night terrors of which I have spoken.

There are also various degrees of disturbance and restlessness at night, which cannot perhaps be fairly classified as night terrors. One of the mildest forms is *grinding of the teeth*. Such cases can be recognised by the fact that the teeth are bevelled down to the same level. At the other extreme is *somnambulism*. All such cases must be treated on the lines already laid down.

The last neurosis I shall describe is enuresis, or *nocturnal incontinence*. I do not pretend to explain how it is that enuresis arises. The exact mechanism of the bladder is not fully understood, but for practical purposes you may compare enuresis to the automatic action of the bladder which you find in many diseases of the spinal cord; and probably it has a very similar explanation. Just as in diseases of the spinal cord, when the brain impulse is cut off by a lesion higher up, the bladder takes to emptying itself automatically when it is full, so in children the brain cortex has sometimes such an imperfect control over the lower centres that the bladder may take on spontaneous action. You may ask at what age the child ought to be able to control its bladder. I think you may say from three years old. You should not therefore talk of enuresis until some time after that.

The *treatment* of the spontaneous passage of urine during the night must consist in the removal of the causes which lead to it. You will find it always well to inquire into the presence of worms, because the irritation of worms in the rectum seems to be a cause which may give rise to enuresis. You should also inquire in the case of boys about phimosis, or preputial adhesions; these also seem to be one of the exciting causes. Further, you should see that these children do not drink much fluid before going to bed, that they sleep on a hard mattress, with few bedclothes; and it is advisable to raise the end of the bed a little, so that the head is lower than the pelvis, and the stress or pressure of the urine tends to gravitate down into the fundus of the bladder instead of pressing on the more sensitive trigone. Here also you will find in a great many cases that partial asphyxia is the cause of the trouble. And adenoids, again, are by far the most common cause of the partial asphyxia. So that in every case of nocturnal incontinence which is brought before you, you should make a point of examining the throat. Lastly, it is well to examine the condition of the urine, to see if it be acid to an undue extent. I think hyperacidity of the urine as a cause of nocturnal incontinence has been exaggerated. I do not think that it is a common cause. If, however, you find uric or oxalic acid crystals, it is well to give alkalies to make the urine less irritating. I have even seen calculus in the bladder mistaken for incontinence, from the irritation and frequent micturition to which it gave rise.

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If you have employed all these measures—attended to the state of the urine, removed worms and adenoids if they were present, given directions that the child should not have fluid just before going to bed, and that it should sleep on a hard mattress and with few bedclothes,—and if you still fail to cure the condition, what shall you do next? You have then to fall back upon drugs. There is one drug especially which is commonly used in the treatment of nocturnal incontinence, and that is belladonna; and there can be no doubt that it is a drug of extreme value if you use it in the proper way. As far as I know, it is the only drug which has a real controlling influence in the disease. There is only one rule to be remembered in regard to the administration of belladonna, and that is *to give it in large doses.* If you do that you do not need to trouble about any other points. Begin with ℥10 of the tincture, given after each meal; try that for a week, and you will probably reduce the frequency of the incontinence at once. You should then mount up to ℥15 each dose, then to ℥25, and then to ℥30, and so on, until you get the incontinence entirely suppressed. I have no hesitation in going up to a drachm of the tincture, even of the new B.P. strength, after each meal,—that is to say, three times a day. It is extraordinary how children will stand belladonna. It is one of the drugs which have far less influence on a child than on a grown-up person. There may possibly be a complaint of slight dimness of vision from paralysis of the eye muscles, or of dryness of the throat, during the administration; but I have not seen more than once or twice any general disturbance, such as a rash or slight delirium. As a rule there are hardly any general toxic effects from large doses, whereas the local effect upon the enuresis is quite remarkable. Now if you have got up to ℥40 of the tincture after each meal, and then the enuresis ceases, how long shall you continue the drug? I think you should do so for at least a month in the full dose, and then discontinue gradually; climb down again in the matter of doses just as you climbed up. You will then find that in the majority of cases when you have stopped the medicine, the habit has been broken, and the child may be regarded as cured. Other drugs are sometimes recommended as substitutes for belladonna. Ergot has some supporters; strychnine has some; and so has cantharides. I have tried all of them, I think, and I can only say that I am by no means certain that any is of much use. I should say that if you fail with belladonna you may regard yourself as at the end of your therapeutic resources so far as drugs are concerned.

I think you will find nocturnal incontinence of urine is much more easy to cure in boys than in girls. I do not know why that should be, but I think the most intractable cases one has to deal with are in girls. In such cases treatment by local applications of nitrate of silver to the urethra has recently been recommended, a strong solution being applied on more than one occasion. I have seen one case at least where this method effected considerable improvement; but my experience of the treatment has not been great. Still it is one which it is well to have in reserve in the case of girls, for whom alone it is intended.

Finally, I would warn you that you must see that these children are not punished. A case which has recently excited much attention in the newspapers—the Penruddocke case—was an instance in which the mother punished the child because it suffered from nocturnal incontinence. It never does any good, or hardly ever, to do so. Very few cases are due to carelessness on the part of the child; the disorder is due to a want of control of the lower centres by the higher, and the lack of control is not a voluntary one, but purely involuntary. It is a case in which the child cannot exert the will, and therefore it is not only useless, but it is extremely unfair and unkind to punish children for this unfortunate weakness. On the other hand, by carefully following out the treatment I have described, I think it will be your experience that in the majority of cases you can get the patient well.

Scarlatina after Burns.—Leiner ('Jahrb. f. Kinderhk.,' vol. lvi, No. 6) has observed three cases of true scarlet fever in children æt. 15 months, 3 years, and 4½ years respectively, in whom the disease developed a few days after severe burns or scalds. It would seem that there is some relation between burns and scarlatinal infection—whether it be that the burn makes a direct point of entrance for the scarlet fever poison, or whether it only increases the predisposition to scarlatina.—*Amer. Journ. of Obstetrics*, May, 1903.

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SOME ETIOLOGICAL FACTORS OF RHEUMATOID ARTHRITIS.

By R. LLEWELYN JONES, M.B.Lond.,
Late Medical Officer, Royal Mineral Water Hospital, Bath.

THE etiological factors that apparently pave the way for the incidence of rheumatoid arthritis are of the most varied nature, and this want of uniformity strikes one forcibly when examining into the previous histories of rheumatoid patients.

Looking at the disease from the bacteriological point of view, we find the same inconstancy reflected in the number of micro-organisms which are to be met with in affected joints. While, on the one hand, competent authorities state that micro-organisms are present in some cases in the joints, a still more important point to ascertain is where the primary entrance of germs is effected; and this would seem to be best attained by a careful inquiry into the previous history of patients, and by the noting of any clinical symptoms which may serve as interconnecting links between rheumatoid arthritis and some pre-existing or intercurrent malady.

Amongst the many diseases that may pre-exist or run their course side by side with rheumatoid arthritis are those pertaining to the alimentary canal—oral sepsis, chronic dyspepsia, gastric ulcer, and dilatation of the stomach; likewise chronic diarrhœa and ulceration of the intestinal canal.

Hunter has shown us the grievous evils that may follow oral sepsis; and the relationship of tetany to toxæmias of gastro-intestinal origin is also worthy of note, as favouring the possibility that some cases of rheumatoid arthritis may own a similar origin.

The following cases are examples which seem to point to some obscure relationship of this nature:

E. G—, female, æt. 36, single.

Personal History.—Had scarlet fever and measles in early childhood; had chorea when fourteen years old; rheumatic fever at twenty-one years of age, and was then confined to bed for over six months. Eight years afterwards she became anæmic; this was followed by severe indigestion, accompanied by vomiting, the latter at one time being so bad as to necessitate rectal feeding. During this period of gastric trouble she had several abscesses around her teeth, all of which, save two, were extracted. Ever since this illness she has suffered, off and on, with indigestion, vomiting, and diarrhœa. In addition she was troubled greatly with "cramps;" these were very painful, and affected her arms, legs, also muscles of neck. These muscular spasms, when they occurred in the legs, sometimes caused dorsi-flexion of the ankle, at other times plantar flexion, preventing any attempt at walking. At other times the plantar muscles were affected, causing strained position of toes. Her hands during the paroxysms assumed a position of semi-flexion, and when the neck muscles were involved the head was rotated and flexed to one side. Coincidently she suffered with recurrent asphyxias of her extremities, especially hands. Last September twelvemonth her gastric symptoms—vomiting, diarrhœa—became very much aggravated, and, simultaneously, the muscular cramps and vaso-motor phenomena. Her joints now gradually began to swell—fingers, left elbow, ankles, and shoulders,—and for treatment of which she was sent to Bath. On admission she was seen to be suffering from rheumatoid arthritis, with the typical fusiform enlargement of the finger-joints, much muscular wasting, also fine tremor of limbs. Her deep reflexes were very brisk, both in upper and lower limbs, and Chvostek's symptom was easily educible over both ulnar nerve-trunks. During her stay in hospital she suffered from vomiting and diarrhœa, and her joints became worse coincidently, and amelioration of the gastric symptoms was followed by a diminution in her joint troubles. Heart and lungs were apparently normal. Pulse-rate was 80 per minute.

M. A—, female, æt. 23, single, domestic servant.

Personal History.—When a child she had measles, and occasionally sore throats. Since eighteen years of age she has suffered with anæmia. The anæmia became very much worse during her twentieth year; she could hardly follow her occupation as a servant—had to sit halfway on the stairs owing to shortness of breath. Amenorrhœa for three months at a time was present; constipation was marked. During this period she suffered severely with indigestion; pain, shooting through to back, accompanied by severe vomiting; the vomiting was very troublesome for three months. About this time she noticed that her index, middle, and ring fingers used to go "dead white" from time to time. The change in appear-

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ance was accompanied by "cramp" of same fingers, which used to become fixed in a position of semi-flexion. These seizures were most marked in the morning, were provoked by exposure to cold; they recurred daily, sometimes more often. During same period she noticed her feet used to go blue and cold. Swelling of the joints of fingers and puffiness of dorsum of feet gradually supervened. Other joints—right elbow and temporo-maxillary—were subsequently involved. The vomiting, etc., above referred to continued off and on for two years, and is still present, though less severe and less frequent, and any increase in severity of gastric symptoms is followed by exacerbation of joint troubles. She was admitted to the Mineral Water Hospital for relief of her joint condition, which presented the usual characteristics of rheumatoid arthritis. There was, in addition, much muscular wasting, also fine tremor of limbs. The deep reflexes in upper and lower extremities were increased, likewise the jaw reflex, and tapping of nerve-trunks produced contraction of the muscles supplied by them. The vaso-motor phenomena above referred to were well marked and recurred from time to time. Her teeth were extensively carious; her bowels constipated. Nothing abnormal was found in heart or lungs. Her urine contained no albumen.

M. B—, æt. 24, female, single.

Personal History.—Measles, sore throats. When nineteen years of age became very anæmic; this condition of anæmia recurred three times. About two years ago she began to suffer with indigestion, also with abscesses round her teeth. She had eleven teeth extracted, but no false ones were inserted. Her indigestion still continued. Two months afterwards she noticed her feet became very "stiff," with pain in soles, and she suffered with chilblains on toes and heels. Her hands at the same time used to go "blue," and gradually swelling of her joints ensued; and she now presents the characteristic joint swellings of rheumatoid arthritis, affecting the temporo-maxillary joint, as well as those of the limbs.

E. C—, female, æt. 36, single.

Personal History.—Has suffered for the past six years with gastric ulcer. Hæmatemesis supervened on several occasions. During this period she experienced "pins and needles" in her hands, which used to go "dead white" at times, and also was troubled with "cramps" in hands and feet. Last July her gastric troubles increased in severity; hæmatemesis occurred, and the symptoms above noted became much more severe and frequent; and during August joint swellings developed, and in the autumn she was admitted to hospital for treatment of the joints, which presented the usual fusiform type.

E. R—, female, single, æt. 21.

Personal History.—Four years ago she suffered with anæmia, also ulceration of the stomach, with hæmatemesis. The next symptom noticed was that her big toe used to go "blue and white often," and then swelled. The same sequence was observed in opposite toe; afterwards both ankles swelled, also knees. Her hands went through the same changes. She at this time noticed that she "trembled" a great deal. About twelve months ago she noticed her collars getting "too tight," and she suffered with palpitation and feelings of "heat," and causeless diarrhœa. For the relief of her joint condition she was sent to the Mineral Hospital, and presented, in addition to rheumatoid arthritis, the phenomena of Graves' disease. Her thyroid gland was enlarged, her pulse-rate was 100, with slight tremor proptosis, von Graefe's sign, and highly arched eyebrows.

In reviewing these cases etiologically we cannot, at any rate, exclude the possibility that the joint conditions were related in some obscure way to the gastric disorder; and the fact that the two conditions varied concomitantly suggests some interdependence. Such interdependence has been shown to be extremely likely, if not actually proven, in the case of tetany. Noting the similarity of the etiological factors, and coupling with it the fact that periarticular swellings and vaso-motor disturbances may occur in tetany, one is tempted to question whether the muscular cramps and vaso-motor phenomena, complained of by these patients prior to the appearance of periarticular swellings or spindle joints, are not closely allied to the milder and more benign forms of tetany; and that, like them, they are the manifestations of a toxæmia acting through the spinal cord and increasing its reflex excitability.

With regard to these vaso-motor phenomena, which are such prominent features in rheumatoid arthritis, I would like to draw attention to the fact that these blue-white or dead fingers often act, as it were, as intervening links between rheumatoid

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arthritis and some previous disorder. These phenomena are met with in gout, renal disease, malaria, etc., diseases in all of which we have in common an altered blood condition.

If we wish for more direct evidence of their value as signs of toxæmia, we have it indicated by their sudden appearance after intestinal obstruction, Again, tingling, numbness, and "dead" fingers are met with in pernicious anæmia.

Lately I have had under my care a patient suffering with chronic intestinal obstruction from intra-pelvic growth, in whom they were marked features. So frequently have I seen them act as forerunners of rheumatoid arthritis that I think their appearance after acute illnesses, *e.g.* influenza, rheumatism, oft-recurring sore throats, puerperal disorders, etc., should arouse suspicion and direct attention to the general health of a patient; and moreover, if any local morbid condition be present, its persistent and radical treatment should be undertaken. Even when the joint conditions have manifested themselves, local treatment of any intercurrent morbid condition seems indicated, *e.g.* extraction of carious teeth, stomach lavage in gastric disorders, intestinal irrigation in chronic diarrhœa.

Again, the leucorrhœal discharges, so common in these patients, often date from some previous severe confinement or abortion, and here again it seems reasonable to urge local treatment, *e.g.* vaginal douching, curettage.

Such local treatment has been tried in some cases, and with benefit; and its more general adoption might throw much light on the nature of the morbid processes which underlie this serious malady. That thermal treatment, massage, electricity, etc., are invaluable and indispensable adjuncts is undeniable, but they are, after all, only ancillary, and their chief value lies in correcting the evil results of the disease; and only incidentally do they attack the cause of the malady, by improving the general health and thereby raising the *vis resistens* of the individual.

In conclusion I would record my gratitude to the honorary physicians of the Mineral Water Hospital for kindly allowing me to see their cases.

THE sixth edition of what is colloquially known as 'Lawson on the Eye,' published by Messrs. Smith, Elder & Co., thoroughly deserves the wide attention it has attracted, not only for its intrinsic merit, but also for the scholarly and literary manner in which the volume has been revised, and in great measure re-written, by Mr. Arnold Lawson.

Mr. Arnold Lawson tells us in his preface that his endeavour has been to present a work on the diseases and injuries of the eye, with their medical and surgical treatment, which would serve not only for the student of ophthalmology, but would also prove of use as a book of reference for the general practitioner, who is more concerned with the practical relief of disease than with any mere theories connected with it. Mr. Arnold Lawson can rest assured that he has most successfully and ably fulfilled his task. It is to the general practitioner that the book will appeal, because so much attention has been devoted to clinical features and treatment. No one can fail to be struck with the enormous care that Mr. Lawson must have expended on this publication, especially in crystallizing down the practical work of the subject in such an inimitable way. To those readers who remember the last edition, which was published in 1885, the many new features of the work will be very welcome; they will recognise that clearness and simplicity have been attained by dividing the book up into chapters, the chapter on each structure being headed by a brief anatomical outline which greatly facilitates the study of the diseases of that particular structure. Entirely fresh articles have been furnished on "Elementary Optics," "Development of the Eye," "The Pupil," "Affections of the Eye in Diseases of the Nervous System," and on "Heterophoria." In the chapter on "Affections of the Eye in Diseases of the Nervous System" the difficult question of hysteria is most concisely and intelligibly discussed. A noteworthy portion of the volume is the index, which has for once received the attention an index deserves. It has been arranged on a much more elaborate plan than is usual in medical works, and the space it occupies is amply compensated for by its great convenience and utility.

Another point for which all students will be grateful is the excellence of the printing, for in these days of cheap and nasty typography it is something to be thankful for when a book is sent for notice that can be read with ease and pleasure.

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"GYNÆCOLOGY IN GENERAL PRACTICE."*

By THOMAS G. STEVENS, M.D.Lond., F.R.C.S.Eng., M.R.C.P.Lond.,

Obstetric Tutor, St. Mary's Hospital; Assistant Physician, Hospital for Women, Soho Square.

I HAVE given the title "Gynæcology in General Practice" to my address, because I know from my own experience, and from what men tell me, that to the young practitioner, at least, gynæcology offers more difficulties than any other part of his work. I shall try and show you why a knowledge of diseases of women is difficult to acquire, and to see how these difficulties may be overcome, illustrating my remarks by a few observations on the commoner diseases coming before us in practice. There are many reasons why gynæcology presents difficulties to the man entering practice, or even to the man who has been some years at work. First of all it is extremely difficult for the student to gain a practical knowledge of diseases of women at hospital. A man has ample opportunities of examining most of the common surgical and medical conditions for himself, both in the wards and in the out-patient room; but diseases of women are and must always be on a different footing. It is not possible for more than three men to examine bimanually any one particular case, and where the number of students is large this means that a man has to spend much time merely listening to his teacher, and only occasionally has the chance to examine a case. True, the number of cases available, as a rule, is in direct ratio to the number of students—the largest hospitals and schools commonly having the largest out-patient departments,—but this does not amount to a sufficient number to afford frequent examinations for all. Again, the number of patients attending the medical schools' gynæco-

* Presidential Address to the Æsculapian Society, given at the Metropolitan Hospital, October 3rd, 1902.

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logical departments is not nearly so large as it might be, because of the presence of the students themselves; patients very naturally object to many examinations, and so go to the special hospitals where there are no students. One would have thought that there must be enough gynæcological cases in London to keep at least three days a week full at every medical school, but it is not so; there are only two days a week set apart at most, if not all, and one of these somehow always has more cases than the other. On the other hand, at the special hospitals, with no students, every day, except perhaps Saturday, is a busy one. Again, the difficulty of acquiring the *tactus eruditus* is often inherent in the man himself; the sensitiveness of finger-tips varies, and the interpretation of sensations felt presents difficulties on this account. This is easily seen when the opinions of three or four men are taken as to the exact condition of the genital organs as ascertained by the bimanual examination; they hardly ever agree as to the size and position, for instance, of the uterus, and ovaries they hardly ever feel at all. To make a vaginal examination should be an almost painless proceeding, but some men always cause a certain amount of pain, and so never get a chance of making a diagnosis, because the patient strains and resists. To be able to make any kind of examination painlessly is important in our work, but in gynæcology it is doubly so on account of the necessity for absolute relaxation of the abdominal muscles. In hospital work every new patient is examined as a matter of course, and expects it, unless there is some particular reason against it; but in private examinations are often objected to, especially where the practitioner is young, and so, at the very time when he wants most practice, difficulties are thrown in his way, both of diagnosing and treating his patients. I am aware that this is not always so, particularly among educated classes, but refused examinations are heard of sufficiently often to be quite sure that they play a great part in making gynæcology difficult in general practice. It is only necessary to turn our attention to the deplorable statistics of operations for uterine cancer to realise that the impossibility of cure is often the result of the delay caused by a refused examination, and to see that it is the obstinacy of the patient which makes the difficulty for the practitioner. How great this obstinacy is on the part of womankind is often seen in the mother of ten children perhaps, who one would have thought was used enough to vaginal manipulations, and yet refuses an examination, even if told her life may be in danger.

Lastly, the student encounters difficulties in his attempts to assimilate gynæcology from text-books, on account of the apparently mysterious nature of many of the diseases he reads about. Until quite recently the pathology of many of the diseases of women was but imperfectly understood, and widely different views on the subject were expressed, leading to indescribable confusion in the brain of the reader. The fact is that there is no mystery about gynæcology; the diseases of the female generative system are subject to the same laws as diseases elsewhere in the body, and when once understood can be treated on a sound pathological basis. To take a familiar example, endometritis. This, we are told, is an inflammatory disease of the lining of the uterus, and then we are mystified because the author gives drawings of what is apparently a diffuse new growth affecting the whole endometrium; in other words, an adenomatous transformation. How can we reconcile these pictures with the previous statement? Quite easily; but I know of no text-book which does so, and therefore the average student is mystified. Inflammation consists of two processes—an infiltration of tissues by leucocytes under irritation, and an overgrowth of the fixed cells of the part. In one case the first is in excess, as we see in an acute abscess the result of infection; in another case we find only overgrowth present, with no invasion of leucocytes, and this we see very clearly in endometritis. However, there is no mystery about it; some chronic irritation, often bacterial, starts the process, and overgrowth continues. In many instances the chronic irritation is not bacterial, but merely chronic congestion the result of subinvolution or displacement, but is not this comparable to a stage of inflammation? Many other difficulties begotten by text-books might be enumerated, but the one mentioned must suffice. From these observations I think I have shown how difficult it is for the student to acquire a good practical knowledge of gynæcology.

Of great importance in the diagnosis of a difficult case is the history, and here we have to reckon with the patient herself. There is a want of exactness in woman's attitude in general which

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makes it very difficult to get a concise history of her case, and this, coupled with an inherent tendency to exaggerate, often conspires to mislead us. It is no uncommon thing among the lower classes of life for women to describe the most ordinary stomach-ache as "agony," and at the same time to have no idea whatever of the date of their last menstrual period. The first we get into the habit of discounting, but the second may lead us into serious difficulties in diagnosis. The various agencies and accidents to which patients ascribe their troubles often prove stumbling-blocks, and in no case is this more misleading than in the elucidation of a case of amenorrhœa, especially if the patient happens to be single. It is not that such patients have any desire to mislead, but with the fear of pregnancy before them they cast about for any trivial accident or ailment they can think of which has happened to them. In such a case we have no choice but to make a carefully conducted examination, and to absolutely disregard any explanation which the patient suggests is at the bottom of her trouble. It is a great art to seize the important point in a long rambling story, and from it to gradually draw out a clear history, always avoiding leading questions.

The really important points we have to elucidate in any gynæcological case are well known to all of us, but for the sake of argument I will recall them:

The age at which menstruation appeared.

Its amount, duration, and average interval.

The date of the last period.

The number of confinements and miscarriages, and the date of the last pregnancy.

In difficult labour, the character of previous confinements.

The presence or absence of vaginal discharge.

The presence or absence of pain, and its character if present.

Finally, such general conditions as digestion, defæcation, and micturition are all important, for their treatment alone may clear up many a supposed gynæcological case.

If these can be satisfactorily made out, most obscure cases after examination will without much difficulty be narrowed down, and if an actual diagnosis cannot be made, at least the line of treatment will be clearly indicated. Examples of the importance of histories could be multiplied indefinitely, but one that came to my notice recently will suffice. A married woman who had had a child eighteen months previously was admitted to hospital with an abdominal swelling as big as a six months' pregnancy. This had, indeed, been diagnosed as pregnancy with some abnormality of the cervix. It was said that fœtal parts had been felt, but there was some doubt about the fœtal heart. The patient complained of nothing in particular. On inquiry I found the patient had menstruated regularly up to the time of her admission to hospital, and had not had one single symptom of pregnancy except the abdominal swelling. The swelling on examination gave all the signs of a cystic tumour of the ovary, and the uterus could be felt apart from it unenlarged. I removed the tumour, which turned out to be a large dermoid of the right ovary. It may be admitted that an occasional woman menstruates throughout the period of gestation, but how infinitely rare is such a condition! and how many of us have seen an authentic case? I, personally, have not seen one. Practically, then, the deduction is that with normal regular menstruation only a very occasional woman could possibly be pregnant. It is otherwise with the woman who menstruates once or even twice after conception has occurred; no doubt all of us have seen such cases. On the other hand, the converse is often true, that a married woman who misses one, two, or more periods is almost certainly pregnant, provided she is otherwise healthy and not near the menopause. Cessation of menstruation in healthy women is rarely mysterious; some real cause for it can usually be found, and pregnancy is the commonest. Let it be quite certain, however, in making such a statement, that the patient is really "otherwise healthy." Heart disease, anæmia, Bright's disease, tuberculosis, poisoning by lead, mercury, or morphia, will all produce amenorrhœa, but are generally quite obvious causes.

To these remarks on the difficulties of diagnosis I now propose to add somewhat on treatment, taking the more common diseases of women as they occur to my mind, and treating them not as an operating gynæcologist, but as one usually treats them in the out-patient room, or as they would be treated commonly in general practice. As an operator, naturally I feel that many cases one treats medicinally and dietetically would be much more rapidly improved by some operative

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procedure, but in hospital out-patient practice, just as in general practice, there are objections to operative measures, however trivial. Most patients with minor complaints would rather have prolonged medicinal or other treatment than subject themselves to operation, if some improvement, if not actual cure, can be thus attained. Many cannot afford the time to lie up even a week or two; the question of expense has to be considered, and most have rooted objections to operations if they can be dispensed with. So that we all have perforce to treat cases, say of dysmenorrhœa, endometritis, erosions, displacements, etc., medicinally, while feeling that some operative measure would produce a much more rapid and certain cure.

Amongst the complaints which most commonly come before us, dysmenorrhœa in its various forms is pre-eminent. We have no difficulty in diagnosing painful menstruation, but we are often sorely put to it to assign a cause and supply treatment which is of any avail. It is easy to write a long list of causes of dysmenorrhœa, but can we not narrow these down within limits which will indicate the line of treatment at once? I think we can. Broadly speaking, the time of onset of the pain gives the clue. Those cases in which the pain precedes the flow by hours or days have pelvic congestion for their cause, while those in which the pain begins with and continues during the flow have painful spasmodic contractions as a cause, or some *mechanical difficulty of expulsion* of the menstrual blood and *débris* from the uterus. Note I do not say that there is any actual obstruction in the sense of stenosis of the canal; this may be so, but is not proved. The difficulty of expulsion may rest with the uterine muscle. In the first class of case the pelvic congestion may be merely physiological, or may be dependent on inflammatory disease of the uterus, tubes, or ovaries. Whatever the cause, we must relieve the congestion to prevent the pain. In the simplest cases the congestion is the physiological influx of blood always present during menstruation, but worse than usual owing to constipation or chronic dyspepsia. These form a good number of our cases, and are greatly relieved by purgation and antidyspeptic remedies. Personally I always combine epsom salts and bismuth for these cases, the dose of the former depending on the amount of constipation. When dyspepsia is not present extract of cascara gr. ij or gr. iv in pill form is a useful mild purge. In cases where the pain is acute just before the flow commences, hot hip-baths give relief, and as most of these patients are better if they vomit I sometimes suggest an emetic of hot water with a little mustard. Not a pleasant remedy this, and not to be indiscriminately recommended; but given at the right time to sensible patients it often acts in a wonderful way. If the congestion is due to old inflammatory disease of ovaries or tubes, relief may be attained by the use of glycerine plugs or 10 per cent. ichthyol pessaries used every other night and combined with hot douches night and morning. These hot douches to do any good must be copious, two quarts of water as hot as the patient can bear being allowed to run through the vagina slowly. A pint douche run through quickly acts more like an irritant, in the same way as plunging the hands into hot water irritates them, but prolonged immersion soothes feelings of congestion. It is the length of time taken in allowing the hot water to flow through which really gauges the amount of good likely to ensue.

In the second class of case, where painful spasmodic contractions of the uterus occur *during* the flow, we must naturally see that there is no constipation, or dyspepsia, or anæmia, and must give drugs to relieve the pain. Morphia must be avoided on account of the danger of rendering its use habitual. Such drugs as antipyrin and phenacetin in 10-grain doses often do good, but it is well the patient should not know what she is taking. Freshly prepared tincture of cannabis indica sometimes gives relief, but its action is capricious, and some people are apt to be strangely affected by the drug even in pharmacopœial doses.

We must admit that these latter cases often are very resistant to medicinal treatment, and are the ones for which some operation usually becomes necessary. Dilatation of the cervical canal is useless, but if combined with thorough curettage and slitting the cervix, often a cure will result. The cut cervix to be really useful must be sutured by one of the methods which prevents union of the cut edges. Finally, there are cases which seem mixtures of the two preceding kinds, and these we have to treat also by a judicious mixture of anti-congestive and antispasmodic remedies.

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Displacements of the uterus are perhaps the next most common conditions we are called upon to treat, and how troublesome they are! But they need not necessarily be difficult to *treat*. Mark I do not say *cure; that* is always difficult and often impossible. The importance of displacements depends on the symptoms they give rise to, so we may eliminate a large proportion of them because they give *no* symptoms. How common it is for a patient to tell us some indefinite symptoms, nearly always a pain in the side; we find her menstruation is normal, she may have leucorrhœa, perhaps dyspepsia, nearly always constipation, and on examination we find a retroverted uterus. Does the displacement cause these symptoms? In most cases it certainly does not, because simple medicinal treatment will cure them. Replacement of such a uterus, and putting in a pessary, usually does not relieve the symptoms; indeed, it does more harm than good, because it concentrates the patient's mind on her generative organs, is a source of infection, and discharges—and last, but most important, often fails utterly to keep the organ in its place. Retroversion and retroflexion of the uterus, in my opinion, only cause symptoms if the organ becomes congested, or causes pressure on the bladder or rectum by enlarging, as it does in pregnancy. The proper symptoms associated with a congested chronic retroversion and flexion are menorrhagia, leucorrhœa, backache, and possibly frequent abortion. To these we must also add symptoms due to tender prolapsed ovaries. This train of symptoms we must energetically treat, and there seems to be nothing better than rest in bed, glycerine plugs, or ichthyol pessaries, and hot douching locally, with ergot in full doses given internally. When the symptoms are relieved we must take care they do not recur, so we must reduce the displacement and put in a pessary. While on the subject of replacing the uterus let us remember to be cautious in the use of the sound for the purpose. I, personally, never use it, because it is no doubt a most fertile source of infection of the endometrium and tubes, by carrying up organisms from the vagina or exterior. If a case cannot be replaced bimanually in the knee-elbow position, it nearly always means pelvic adhesions, and forcible replacement with the sound will do much harm. Personally, too, I have altogether given up the sound for diagnostic purposes, preferring to trust to my hands to tell me the size and direction of the uterus.

The other class of case in which pessaries do good and may even effect a cure is in a retroversion and flexion occurring immediately after parturition or abortion. Here the organ is soft and mouldable, and, being large, is easily replaced and kept in place by a properly fitting pessary. Many cases of retroversion are congenital, and require no treatment unless they become infected or pregnant. In the latter case, if the patient comes before us early enough, we may be able to replace the uterus and keep it in place by a pessary, and so avoid the abortion which would inevitably occur if left to itself. I have a patient with a congenital retroversion who became pregnant and aborted. Afterwards, although treated by pessaries, the displacement recurred, and I advised it should be left alone, as it caused no trouble; but at the same time advised that I should see her at once if she became pregnant. I saw her the other day; she was pregnant about six weeks. The uterus was easily anteverted and kept so by means of a ring pessary, and her pregnancy is progressing naturally without any unusual disturbance. I believe the success of treatment depends on the fact that the organ is enlarged. Congenital retroverted uteri are invariably small and badly developed, and so offer nothing for a pessary to exert leverage on. When they enlarge through pregnancy the very size and weight of the organ helps the pessary in its action, whatever that may be. I do not believe a healthy uterus ever becomes retroverted by a fall or other accident. One often sees cases which are explained by this means, frequently in unmarried girls, and up to now I have not yet seen one which I could believe was caused in this way. All such cases I have seen are apparently congenital, and do not cause the symptoms complained of.

With regard to the so-called anteflexions of the uterus, in my opinion such cases are congenital malformations and in no sense displacements at all, the only exception being the acute anteflexion caused by cicatricial contraction of the utero-sacral ligaments. No pessary whatever relieves the symptoms—dysmenorrhœa and sterility constantly associated with this shaped uterus,—and so I never recommend the use of one. Above all, I never use an intra-uterine stem, which always does more harm than good in the long run. *Prolapse* of

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the uterus, on the other hand, we must always treat, for it tends to become worse, and is attended by pathological results which are incurable. Some kind of support we must use, and in early cases, especially those following immediately upon parturition, even *cure* can often be attained by means of an efficient support, combined with some medicine calculated to procure involution of the uterus and ligaments. Ergot in full doses, combined with iron, and prevention of any straining at stool, is the sheet-anchor of our treatment here. The prevention of these conditions is often in our own hands, because they are nearly always slowly produced after parturition. If we sew up *all* perineal tears by deeply placed sutures, prevent any strain due to cough or constipation, and secure involution of the uterus and ligaments, we shall see very few cases of prolapsus. In old severe cases, which have been neglected, and where the levator ani muscle is quite atrophied, we can do very little. Cup and stem pessaries in my experience are useless; the slightest strain will force them out. What I often do with them is to put in the largest ring pessary possible, and then make the patient wear a firm oblong pad outside the vulva, attached to straps round the waist. The ring alone will not stay in, but the pad outside often enables the patient to retain it. The Zwancke pessary is sometimes useful if there is any part of the posterior portion of the pelvic floor left with sufficient tone for it to rest upon. It must, however, always be taken out at night, as if kept in for any length of time it always sets up ulceration and causes vesico-vaginal fistula. I have recently seen two fistulæ caused in this way, and, curiously, they were both in the middle line. It does not seem possible that pressure alone could do this, for with this pessary the greatest pressure is lateral and posterior. It seems almost as if the metal hinge in the middle line sets up some electrical action in the moist vagina and gradually ulcerates the anterior wall into the bladder.

Of all the symptoms to which middle-aged women are subject, none, I venture to think, gives more trouble than *frequency of micturition*. All classes of women are liable to be thus affected, and the condition used to be spoken of as "irritable bladder." To make a diagnosis of the actual cause of this symptom is extremely difficult, but it must be recognised now that there is no such thing as "irritable bladder." Frequent micturition is only a symptom of some disease affecting the bladder, urethra, or urine. It was not until surgeons began to use the cystoscope freely that the manifold causes of this symptom were found out. Many of the causes external to the bladder itself are easy to diagnose and treat, but those affecting the lining, always the most obscure and treatment-resisting, require the use of the cystoscope for diagnosis, and so do not come within the sphere of all of us. But, however obscure the case and however long standing, we may still do much without the cystoscope, but we must be systematic about it.

The causes external to the bladder are:

Uterine prolapse and cystocele.

Retroversion with pressure on the neck of the bladder.

Pregnancy, by reason of the anteversion it causes.

Uterine growths invading the bladder.

Pelvic peritonitis, causing adhesions to the bladder and so intolerance of distension.

All these are easy to recognise by ordinary methods, and suggest at once their appropriate treatment, except the last. It occurs after pelvic peritonitis, and the adhesions formed cause great pain when the bladder distends, probably because of the stretching produced. Tolerance of distension can only be established by daily injections of non-irritant fluid, more and more being used each day until the bladder becomes tolerant of a moderate distension. A tedious process, truly, but one attended by gratifying results when carefully carried out.

The causes situated in the urine itself can be diagnosed by simple analysis. They are:

Bacteriuria.

Lithiasis.

Oxaluria.

Hyperacidity and excess of phosphates.

The first, diagnosed by the microscope, may be often cured by giving urotropin in 7-grain doses three times a day.

The others may generally be cured by careful dieting, and medicines calculated to render the urine alkaline. Citrate of potash with hyoscyamus and camphor water is the prescription most generally useful, but where there is great acidity of urine and phosphatic excess, boracic acid in 10-grain doses is often useful.

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Of the *urethral* causes, caruncle is self-evident, and urethro-cystitis generally can be easily diagnosed; but where the bladder lining is affected we can only diagnose by a process of exclusion, and differentiate between them by means of the cystoscope. The chief of these are:

Vesical hyperæmia.
Catarrhal cystitis.
Cystitis with ammoniacal urine and pus.
Vesical papillomata.
Vesical varices.
Vesical growths.
Tubercle of the bladder.
Vesical calculi.

Vesical hyperæmia and catarrh are not attended by pus-formation, and if diagnosed may be cured by daily irrigation with boracic acid lotion and instillation of 0·25 per cent. solution of silver nitrate. Cystitis with pus-formation we are all familiar with, and is often difficult to cure; but frequent washing out or even drainage of the bladder, together with urotropin internally, will often do much good. The other conditions require operative treatment, and scarcely come within the scope of general practice.

The subject of uterine inflammations, corporeal and cervical, is one upon which much could be said, but I will content myself with the remark that I suppose we are all agreed that the days of caustics are past. We no longer apply chromic or other acids week by week to cervical erosions, but either thoroughly remove them with one application of the knife or curette, or treat them by astringent douches. Very good results, too, can be attained by the latter simple method, provided it is continued long enough and the patient's general condition is attended to at the same time. The old method of treating these conditions by caustics only resulted in increased adenomatous growth, unless the caustic was strong enough to at once destroy everything down to the uterine muscle.

The differential diagnosis of abdominal tumours is a most fascinating subject, but I think I have sufficiently indicated the lines upon which this can be safely approached, and if an absolute diagnosis cannot be arrived at we can at least narrow our views of any particular case down to the point at which the line of treatment becomes clear.

Illustrations of the importance of diagnosis and methods of treatment in gynæcology might be amplified until one had inflicted a whole textbook upon you. But this is not my purpose to-night. I do not wish to pose as a teacher or to impose my views as the only correct ones. My wish has been to take a few examples of the common problems in diseases of women which come before us, and to indicate the lines of thought by which we can approach, diagnose, and treat them with some showing of common sense. In conclusion, let us be quite clear on a few points. Gynæcology is no longer a mystery, but is governed by the same laws which pertain to medicine and surgery generally.

The treatment of diseases of women may be medicinal or operative. The first, used with discrimination, will enable us to successfully treat a large proportion of our patients, while the second has nearly always clear indications and must be always carried out with the strictest regard for technique. Let us avoid pessaries in all but most carefully selected cases, and above all avoid the tinkering applications which at one time were so much in vogue in the treatment of minor diseases of women. I am aware of the shortcomings of my address to-night, but I have put before you in all sincerity a few thoughts which often pass through my mind while at work.

CLINICAL LECTURES
ON
DISEASES OF CHILDREN.

LECTURE XV.

ON MENTAL DEFICIENCY IN CHILDHOOD.

Delivered at the London Hospital, December 16th, 1902.

By ROBERT HUTCHISON, M.D.Ed., M.R.C.P.,
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GENTLEMEN,—Our subject to-day, which will close this course of lectures, is the mental deficiencies of children. This is a subject which deserves your very closest attention, because you will find when you go into practice that mental deficiency is not uncommon in children, and you will frequently be consulted about it. Moreover it is a subject in

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regard to which if you make a mistake you are likely to get into discredit. The frequency of mental deficiency in children you will realise when I tell you that about 1 per cent. of all the children of school age are the subjects of it. But before I go further into this matter I want to define for you the use of terms in regard to mental deficiency. You will find, for instance, such words as idiocy, imbecility, feeble-mindedness, and backwardness all used with regard to children whose mental development is defective. At the present day the use of the first three of these terms has been practically discontinued, and the expression mental deficiency is used in place of them. But if you are going to adhere still to the use of the term idiocy, one would reserve it for the lowest grade of case,—that is to say, those in which the mental impairment is of the extremest form. Above those, somewhat better in their mental development, will come imbeciles, and higher still, nearer to the normal children, will come those who can be called the feeble-minded. Backwardness is a thing to be sharply distinguished from mental deficiency, and therefore we shall still retain that term, and I shall define what is meant by it later on.

Coming closer to our subject, it will be convenient to look first at the general diagnosis of mental deficiency; secondly, at the clinical classification of cases of this affection; thirdly, at the etiology so far as we know it; fourth, at the prognosis; and fifth, at its treatment. I shall begin with the diagnosis.

When you wish to diagnose a mental condition, even in an adult person, it is well to put to yourself three questions: what does the patient say? what does he do? what does he look like? In diagnosing mental deficiency in children you will find that these three modes of attacking the problem will be of great service to you.

First, then, what does the patient say? Speech, of course, is the great mode of intellectual expression; it is one of the things by which you can judge best of a man's intellectual development. In the case of children who are mentally deficient you will find often that it is not so much what the patient says as what he does *not* say, which is helpful to you and which will arouse your suspicion. You will remember that by the age of three a child ought to have learnt to talk fairly well; and if a child is brought to you at that age who is still unable to talk, you may conclude either that you are dealing with a condition of mental deficiency, or that the patient is deaf. There is a third possibility, but so uncommon as to be scarcely worth considering—namely, that the child may be the subject of what is called congenital aphasia. But that is very rare. So practically a child of three years of age who cannot talk is either deaf or mentally defective. As regards what one may call *qualitative* alterations of speech, I would only point out that you must not conclude that such peculiarities are necessarily signs of mental deficiency. Take such a thing as *stammering*. That is by no means an indication of mental weakness; indeed, subjects who stammer are often above the average of intellectual development. There is another condition termed *idioglossia*, in which the child talks in a sort of gibberish of its own which is entirely unintelligible to other people, and that also, although you may think it is an indication of mental deficiency, is not so. These children are often otherwise intellectually sound. On the other hand, the condition of speech called *lalling* is certainly suspicious of mental deficiency, and another peculiarity certainly exhibited by some defective children is *repetition*; they have a habit of repeating after you the question which you ask them, or the things which you say.

Next let us consider what the child *does* which arouses your suspicions. I would point out that one great sign of mental deficiency is an inability to perform at the proper times complicated co-ordinated movements. The child will therefore be late in passing many of those "milestones of development" of which I spoke in one of the early lectures. He will be late in holding up his head; instead of being able to co-ordinate the muscles which hold the head on the trunk by the third month, he may not be able to do so until he is a year old, or possibly more. He will be late also in sitting up; instead of being able to sit up at the end of the sixth or the ninth month, it will perhaps not be until the eighteenth month that he can do so. The child will also be backward in walking, and, as we have just seen, in performing those co-ordinated movements which give rise to speech. Further, mentally deficient children are apt to fail in the power of grasping things; they are wanting in the power of *acquisitiveness*, as it has been said, for grasping involves complicated co-ordinated move-

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ments by the muscles of the arms, and so you are not surprised to find that it is a function which is acquired unusually late by children whose cerebral development is defective. There will also be a lack of co-ordination in the muscles which move the eyeball and which enable the child to look fixedly at any object. Hence the gaze is vacant.

Amongst positive motor signs which will arouse your suspicion is the performance of rhythmic movements. These are a common indication of mental deficiency. For instance, there are swaying movements of the body, jerking of the shoulders, or grimacing movements. Such movements as those, constantly repeated, should arouse your suspicion with regard to the mental condition of your patient. There may also be certain tricks of movement which are suspicious. I refer to such things as hand-sucking, and the making of meaningless gestures. Great restlessness is also a common sign. This often goes with sleeplessness at night, and that again with apparently causeless screaming or crying.

We now come to what the patient *looks like*. The facies or *facial expression* of the child is, after all, perhaps the most valuable index which you have of the mental state. I told you that the face in children is a mirror of the mind in a way that it is not in grown-up people, and there is no class of case in which that is of greater service to you than in the mentally defective. So much is this the case that many forms of mental deficiency in children have an absolutely characteristic facies, so that you can say at once this child is mentally deficient and belongs to such and such a class. We shall see immediately when we speak of classification that one makes large use of this in the subdivision of cases of mental deficiency into different clinical groups. But apart from those which have a characteristic facies you will find in almost all mentally deficient children *something* in the expression which will arouse your suspicion. There is a vacant look, or perhaps grimacing, or an inability to recognise the mother or the nurse. All these things should put you on your guard. You will also find that the size and shape of the *skull* is often of great service to you in enabling you to arrive at a diagnosis; because, just as in facial expression, so here, with regard to the skull there are abnormalities which are absolutely characteristic. Then you will find in looking more carefully at such children that you are apt to get what are spoken of as the *physical stigmata* of intellectual defect, which have been described by Dr. Warner under what he terms "the law of coincident development." This simply means that if one part of the body is defectively developed, other parts are apt to suffer also. If, for instance, the child has a badly developed brain, it is more likely than other children to have congenital lesions of the heart, or to have supernumerary digits. Amongst the physical stigmata of mental deficiency are what are termed marked epicanthic folds,—that is to say, folds of skin which come down from the upper eyelid to the lower and cover over the caruncle. A high-arched or Gothic palate is another common physical stigma; and it frequently goes with narrowness of the naso-pharynx and bad development of the jaws, and therefore also of the teeth. Then you will find adherent lobes of the ears more commonly in mentally deficient children than in others, the lobe being tacked on to the skin of the cheek instead of being free. You find, too, not uncommonly, congenital heart lesions, supernumerary digits, harelip, cleft palate, and opacities of the media of the eyes. In a doubtful case you will look for such physical stigmata, and the finding of them will be an additional point in favour of the view that the child is mentally defective.

We next come to the second division of the subject—namely, the *clinical classification* of cases of mental deficiency in children. One would prefer to use a pathological classification if that were possible, but at present it is not. The pathology of many cases of mental deficiency is still quite obscure, and you will find it, as a practical matter, more convenient to group the cases according to the chief clinical symptoms and features which they exhibit. We shall begin with those cases which present a marked facies.

1. The first group of those are the *cretins*. It is extremely important that you should be able to recognise the cretin, because they form that group of mentally defective infants which are the most amenable to treatment; and it will depend upon your being able to make the diagnosis within the first year of life whether the child will grow up mentally sound or whether it will be considerably defective. The recognition of the cretin in its early stage while it is still young is not always easy, and the classical pictures of cretins which you see in books are

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usually taken, unfortunately, from children several years old, by which time, of course, anyone can make the diagnosis; but if you recognise them only then, you recognise them too late to do anything of service to them. In order to do a cretin real good you have to catch him early, and if you do so, and treat him properly, he will grow up into a child which is practically indistinguishable from a healthy and normally developed one.

What, then, are the features by which you will recognise cretinism in its early stages? Before I reply to this I would say that the cretins are not so common as is supposed. There is too great a tendency to label almost every mentally defective child a cretin. I would impress upon you the fact that cretinism is a rare condition. Out of seventy-five mentally deficient children of which I have notes, only 6 per cent. were examples of cretinism; whereas Mongolism, which it is very apt to be mistaken for, occurs with much greater frequency, amounting to about 36 per cent. of my cases.

With regard to the cretin, then, the complexion is parchment-like in tint, the hair dry and scanty, and the skin redundant, so that there is a puckering of the forehead and puffiness below the eyes; there is often a squint, but by no means invariably. There is rarely, if ever, nystagmus. When you look at the shape of the head you find it tends to be long; it is dolichocephalic. On examining the trunk you will find that the skin is dry and rough. There is frequently an umbilical hernia; the hands are short and broad, and the finger-tips square. In manner cretins are dull and lethargic, and a feature which you will recognise in them from earliest life is that they suffer extremely from constipation. I have said nothing of the supra-clavicular pads of which you will read in books, and for this reason: that supra-clavicular pads are not developed, so far as I have seen, until a cretin is at least a few years old; they are not seen in the earliest stage; and, although it is perfectly true that you get them afterwards, yet by that time it is too late for them to be of use in diagnosis. I should perhaps have mentioned as a characteristic feature the tendency to protrusion of the tongue. The tongue is large, broad, and thick, and it is apt to be kept protruded. All those characteristics taken together make up a picture which, if you have once seen it, you are not likely to miss again.

2. Now I pass to consider *Mongols*. The term Mongolism was first applied to this group of cases by Dr. Langdon Down, who was a physician at this hospital, from their resemblance to members of the Mongolian races—the Chinese and Japanese—in face, and particularly because they have the oval, tilted eyes, which are characteristic of these nations. When you come to compare their characteristics with those of the cretins, with which they are apt to be confused, you will find that the skin of the Mongol is smooth and white, not parchment-like in tint, and that they have a good complexion. They have no puckering of the eyebrows and no puffiness of the eyes, the hair is fine and abundant instead of being dry and scanty, and they almost always have a squint, and usually nystagmus as well. The head, instead of being dolichocephalic, is brachycephalic, and the occiput is almost in a direct line with the neck; whereas the cretin has a head projecting back beyond the neck. The finger-tips are rounded, and they have a curious incurving of the little finger. Moreover, instead of being dull and lethargic, they are bright and lively in manner. So you see they contrast in every respect with the cretins, and if you have seen a good example of each of them you need never mistake one for the other. Of the physical stigmata which I spoke of, marked epicanthic folds are a striking feature of Mongolism, and so is congenital defect of the heart. Mongols tend to suffer with great frequency from defects of the septum and other lesions of a similar sort. Out of eighteen cases of which I have notes, five had marked congenital heart disease.

3. I now pass to the third group—namely, cases of *microcephaly*. Those cases, as the name implies, are distinguished by smallness of the head. The normal head measures 13 inches in circumference at birth, and at nine months old 17 inches, 18 inches at one year, and 20 inches at five years; and heads which come below these dimensions may be described as cases of microcephaly. But I want to point out to you that the characteristic of microcephaly is not merely deficiency in the circumference of the head, but an alteration in shape. I shall show you photographs presently indicating that the characteristic point about a microcephalic head is that the deficiency affects chiefly the frontal region and the vault. The base of the skull is apparently as well developed as in health. So a constricted frontal region with a small circumference are the characteristic points. The size of the head may be

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extremely small. The smallest I have seen was in a child three years old in whom the total circumference was 13¾ inches,—that is to say, only ¾ inch more than it should be at birth. One sees them of all measurements, varying from what I have told you to nearly the normal size. Otherwise the child is usually well developed in face and body, but has a dull demeanour, and tends to be apathetic in manner.

Cases of the microcephalic type are not at all uncommon. They made up 26 per cent. of my own cases; and from what I have seen here and at Great Ormond Street Children's Hospital I think the condition tends to occur with unusual frequency in children of Jewish parents. That is quite the reverse of Mongolism, from which condition the Jewish race seems to have an immunity rather than otherwise.

4. Opposed to microcephaly is *hydrocephaly*, in which the head is unusually large. I have said so much about the diagnosis of the hydrocephalic head and the means of telling it from rickets that I shall not go over that again. Hydrocephaly is not, however, a common cause of mental deficiency, and it is extraordinary how large the head may be and yet the mental power be very well retained. I shall show you the photograph of a child whose head was 26 inches in circumference, and who yet conversed quite intelligently. But such children are apt to be backward in their movements, because the large head upsets their balance and makes it difficult for them to walk; but the mental power is retained in a surprising degree, even though you know that the cortex of the brain must be flattened out almost like a sheet of note-paper.

5. We come next to the *paralytic group*, in which there exists some degree of paralysis in addition to the mental deficiency. Those cases belong to that group of infantile cerebral palsies of which I spoke in my last lecture. The children who are most apt to suffer from mental deficiency with paralysis are those in whom there has been a lesion of the brain in intra-uterine life or at birth, and I think it is more common in those who have paraplegia than in those who have hemiplegia. You are apt to arrive at an exaggerated estimate of the mental defect in these children, because, owing to paralysis of muscles, they have a difficulty in getting about and living an active life like other children, and accordingly their education suffers, and to this, no doubt, part of their intellectual dulness is to be attributed.

6. There are two groups of cases of mental deficiency which are *associated with fits*, the eclamptic and the epileptic group. The distinction between these is that *eclamptic imbecility* includes those cases in which a fit has been the starting-point of the mental defect. The child lives for the first two or three years of its life quite normally, and its mental development goes on in the ordinary way, and then it has a severe convulsion which appears to damage the brain, and from that time the mental development suffers although there is no repetition of the fit. In the *epileptic group* the case may start with a fit, but the characteristic feature is that the fits recur; the child goes on having fits, and it is in consequence of the continual fits that the mental state suffers. You may take it that if epileptic fits are frequent in a child below seven years of age, that child is almost certain to have as a consequence mental deficiency. There is nothing which apparently upsets the equilibrium of the brain more than the repeated occurrence of fits.

7. The next group is *idiocy by deprivation*. By that one means those cases which are mentally deficient because the "avenues of knowledge" are blocked. The classical example is that of Laura Bridgman, of whom you may have heard. She was blind and deaf and all her senses were gone, and as long as she was in that condition she was practically idiotic. Very many children suffer in their mental development because of this blocking of the avenues of information; because their hearing or their sight is bad. And whenever you are brought face to face with mental deficiency you have to ask yourself,—may not this be produced by the fact that the child has a difficulty in acquiring information owing to the fact that it cannot hear properly or see distinctly? The commonest causes are middle ear disease, frequently the result of adenoids; and errors of refraction, particularly hypermetropia and astigmatism. As a consequence of these defects the child fails to benefit like other children from instruction at school, and so falls behindhand, and in extreme cases may be regarded as a case of mental deficiency. But many of these cases belong to the group of mere *backwardness*. And that brings me to ask what is to be your distinction between mere backwardness and true

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mental deficiency. You will find it convenient to remember in this connection the dictum of Charles West that a mentally deficient child would be abnormal for any age, whereas a backward child is merely abnormal for its own age. For example, a backward child of six may be at a mental stage which would be normal for a child of five. But a child who is mentally deficient you cannot regard as normal for any age. You will find that useful as a practical distinction.

8. I now come to the next group, which is one of considerable rarity—namely, *primary amaurotic mental deficiency*. This is a group of cases which ought to be familiar to you here, because it was first described by Mr. Waren Tay, I think in the year 1871. It is a group which is marked by certain very definite features. First, the cases tend to occur in families, several members of a family being likely to suffer. Secondly, in the vast majority of cases the children are of Jewish parentage. Thirdly, they are characterised by the fact that the children are normal at birth and for some time afterwards; that then loss of vision sets in, associated with characteristic changes in the fundus of the eye and with paralysis, which leads by about the second year of life to death. Such cases are very far from being common. It is well, however, for you to have them before your minds, particularly in this part of London, where we see so many Jewish patients.

9. Lastly, I come to a great group which you can refer to none of these previous divisions, and which may be described as *unclassifiable* cases. They make up the majority you meet with; they were 32 per cent. of all the cases I have seen. You may call them, if you like, congenital idiocy, but I do not think that helps you much. They are characterised by mental deficiency associated perhaps with some "physical stigmata," and you can only diagnose them by giving heed to the general rules I have laid down—to the speech, general appearance, and behaviour of the patient,—and if you attend to these points you are not likely to overlook the mental defect.

I now pass to consider rapidly the question of the *etiology of mental deficiency* in children. With regard to a few of these groups there is a definite causation. Cretinism, for example, is due to congenital absence of the thyroid gland, but you cannot say of many that there is such a definite causation as that. In the paralytic group, too, you have a fairly definite cause—namely, damage to the brain of some sort, occurring during the act of birth or during intra-uterine life. In the group of deprivation cases you have a definite pathology in the absence or defect of certain organs of sense. In hydrocephaly you have often a history of antecedent basal meningitis. But in regard to many of the cases you can find no definite cause at all. You will find that certain factors are commonly believed, especially by lay people, to play a part in the production of mental deficiency. You will hear it said that these children are often the offspring of intemperate parents; or that there is insanity, or syphilis, or tuberculosis in the family; or that the parents have been related to one another before marriage. As far as I have been able to make out, you cannot say definitely that these factors have any very great influence. Out of seventy-five cases I only found one in which consanguinity was present, and in that case the parents were first cousins. And I cannot find that the parents of mentally defective children are addicted to intemperance more than others; whilst the co-existence of congenital syphilis is quite exceptional.

A cause which might be more likely to produce mental deficiency is bad health in the mother during her pregnancy. There have been experiments on chicks which show that if an egg is incubated at a higher temperature than usual the chick which results is apt to be abnormal and defective in development; and you might suppose that, if a mother has an attack of fever during her pregnancy, so that the incubation of the fœtus proceeds at a temperature two or three degrees above the usual, mental defects amongst others might result. And I do not say that that does not sometimes happen. You will frequently get a history of bad health during the pregnancy, or a history of worry; but you must remember that such occurrences are also extremely common in pregnancies which produce normal children, and you must always allow for a tendency on the part of the mother to assign *some* cause for the mental defect in her child. I do not think it has been clearly proved that any of these things is a constant factor.

I want to speak more definitely with regard to two of the above groups—namely, microcephaly

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and Mongolism. The pathology of microcephaly seems to be an arrest in the development of the brain as a whole. It is not due, as has been sometimes said, to a premature closure of the cranial sutures. You know that a method of treatment was based upon that assumption—namely, craniectomy, artificial openings being made in the skull under the impression that the brain ceased to develop because the sutures closed too soon. As a matter of fact the reverse seems to be the case; the skull closes soon because the brain is not properly developed; so that the operation was foredoomed to failure.

With regard to Mongolism, I think you can trace a fairly definite cause. You will find that Mongols tend to be the last children of large families. It is a condition which results when the reproductive powers are exhausted, especially in the mother. Out of fifteen of my cases of Mongolism, the mother was forty years old or upwards in nine; and in another three the mothers were between the ages of thirty-eight and forty. Dr. Still, of Great Ormond Street Hospital, reports eighteen consecutive cases, out of which ten were the last children of a large family, and in the others the child was the last but one. Why exhaustion of the reproductive power should result in Mongolism I do not know; but there seems to be an arrest at some stage, beyond which it is impossible to carry the development.

With regard to the *prognosis* in mental deficiency, you have to consider it first in relation to the life of the child, and secondly in relation to its mental progress. As regards life, you will find that mentally defective children tend to be physically weak and wanting in resisting power. It is perhaps fortunate that that is so, because the majority of them never reach the age of puberty. If they have an illness they take it badly, and are apt to go down before it. This feebleness is perhaps more marked in Mongols and cretins than it is in others, and the members of this group are particularly liable to be carried off by tuberculosis. You have also to remember in giving your forecast that many of them will be apt to suffer from fits, particularly in the paralytic cases. Of course, those which are epileptic already will always have fits as one of their prominent symptoms. With regard to mental development, there is nothing more difficult than to speak confidently about the future, and I advise you to be always very cautious. You may say of cretins that if thyroid gland is administered from the first year of life they will grow up to be almost normal individuals. You can say with regard to Mongols that in the highest grade they will be fit for physical work, but they will never be able to do much brain work of any sort. The prognosis of microcephaly will depend on the size of the skull; the smaller the skull the more hopelessly idiotic the child will be, and it is only those children in whom the skull is near the normal size that you will have any appreciable mental power at all. With regard to the epileptic cases, you may assert that they tend to get worse rather than better as they go on. Each successive fit leaves the brain in a more damaged condition than it was in before. But the great difficulty in the matter of prognosis is in regard to those unclassifiable cases which make up the last group. It will be well to remember, as has been pointed out by many observers, that the more healthy and natural-looking the child is, the worse is the prognosis as regards mental development. Those cases which look best in physical development often give the worst outlook with regard to mental improvement.

The *treatment* we can dismiss in a word, because, I am sorry to say, for many of them you can do but little. I urge you always to remember, however, that there is a physical side to mental deficiency, and although you can do nothing for the mental development of these children, you can do much for their physical state; you can remove obstacles out of the way of the feeble brain. You can remove adenoids, which make it difficult for them to hear. You can order them spectacles, and make it easy for them to see. You can divide tendons if they are paralysed, and make it easier for them to use their limbs. If the child is rickety, you can treat the rickets. By removing these difficulties you can, especially where there is only slight mental deficiency, make all the difference to the child, and put it upon a very much higher plane than before.

With regard to mental improvement, all you can hope to do is by careful and persistent training. In recent years much progress has been made in this direction, and the lot of the mentally deficient child has never been as favourable as now. Under the Elementary Education Act of 1899, special

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classes have been instituted under the School Board in London for the training of defective children; and much has been done by the Kindergarten methods in teaching these children to use their muscles, and make the best use of the feeble brain-power they have. It is only on those lines that you can hope to do good in those who are improvable. For those who are unimprovable there is no refuge but the idiot asylum, or, for people who are able to afford it, special guardianship.

WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

The Specific Treatment of Infection in Man.

We have seen that a susceptible animal acquires immunity against a given infection during and by recovery from the disease concerned, and that immunity can be conferred upon it either by the injection of an immune serum or by the method of inoculations. The latter process leads to an active durable immunity, whereas the former gives an immunity of only temporary duration, which is entirely passive in its character.

The production of passive immunity by the injection of immune serum, whether as a prophylactic agent or in the treatment of the actual disease, has this great drawback, that the development of active acquired immunity is thereby checked. This happens in the following manner:

The immune body needed and the antibodies of the bacterial toxins are supplied in the injected serum. The individual concerned has, therefore, only to provide bacteriolytic ferment; and since the bacterial products are antagonised by the specific bodies in the immune serum they cannot cause a formation of such bodies by the side chains of the infected individual. Hence no facility in the formation of immune body and of antitoxins is acquired, and accordingly the individual remains susceptible. This represents an extreme case where sufficient immune serum is injected to entirely neutralise the bacterial products, but similar considerations show that any and every injection of the serum must, in so far, oppose the development of the immunity reaction and lessen the immunity acquired.

On this account it is advisable to avoid the exhibition of excessive doses of such sera, and to employ the minimum amount required to ensure the recovery of the patient.

The production of immunity by bacterial vaccines is a production of acquired immunity of active character by the reaction of the individual concerned to mild infection with the specific agent of the disease in question. It is an imitation of the way of nature, and is a truly scientific method of protection. That natural processes continually work along this line is demonstrated by the following facts:

In times of epidemic it is found among persons similarly exposed to the infection that, for every case prostrated by a severe attack, many may have some slight indisposition, and many more exhibit no objective evidence of reaction. Now it cannot be that all these latter have escaped infection where all are equally exposed, and hence we may conclude that in these individuals the natural resistance is at such a level that the repeated entrance, in small quantity, of the infective agent leads to a gradual development of immunity without the production of a marked and visible reaction.

Thus probably must be explained a number of the slight febricula, sore throats, and passing diarrhœas, which present themselves during an epidemic of infective illness, as well as the immunity of those who work continuously in fever hospitals, and the remarkable resistance of pathologists to post-mortem room infections.

The wide-spread prevalence of the natural method of protection is further evidenced by the diminution of susceptibility in general with increasing age, till after thirty or thirty-five many of the specific infections are quite unusual. The process is exemplified in the parallel frequent observation of antirennin in the blood of horses, of anticholera protective bodies in the blood, and cholera vibriones in the *solid* stools in healthy men during a cholera epidemic (Sobernheim), and of diphtheria antitoxin in the blood of uninoculated horses, and of healthy men who have never suffered from diphtheria (Wasserman). Moreover, such occurrence of antibodies in the blood without previous attack of the disease concerned is much more frequently to be observed in adolescents and adults than in young children (Wasserman), thus pointing clearly

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to their origin from the reaction to repeated small infection.

It thus appears most clearly that the inoculation method imitates the way of nature, and like the latter it affords a durable immunity.

The pioneer discoveries of Jenner laid the foundations for the scientific use of this procedure. It is unnecessary here to enter the discussion of the causal agent and relations of variola. That its virus is identical with that of cow-pox which is used for vaccination is rendered highly probable by the work of Klein, of Copeman, and of Hime, and many others. Moreover many of the strains of vaccine lymph now used have been derived originally from variola. The inoculation, therefore, is specific.

In rabies a considerable measure of success has followed the distinguished work of Pasteur. And Wright's inoculation against typhoid fever is of undoubted value. The anticholeraic vaccines used by Haffkine had not a great success. And all the evidence points clearly to the fact that, while an attenuated virus must of necessity be first employed in man, we cannot hope to gain complete protection until by later treatment with more active forms a full resistance to the bacterium of normal virulence has been attained.

The method of inoculation has, however, no value where an infection has already gained maturity, though by its more direct immediate entrance it may be able to induce immunity during the incubation period of the natural disease where this is long enough—as in variola. We therefore need a therapeutic agent, and this we find in the specific sera. In the use of these we seek to give assistance and to facilitate recovery from disease by a transference of antibodies to the affected individual, leaving him dependent only on his own phagocytic ferment to destroy the invading micro-organisms with the assistance of the immune body injected. Toxic bacterial products will be neutralised by the antitoxic bodies of the serum. And Madsen has shown that even toxin which has already attached itself to cells can be removed from them and rendered harmless if a sufficient mass of the free antitoxin be injected.

In diphtheria the results from antitoxin treatment have been more than excellent. In tetanus also numerous cures have been recorded. In severe cases the serum should be injected intracranially where it can act at once, and in considerable concentration on the toxin which has already reached the nervous centres.

A number of other sera have been tried with less success, such as the anti-sera for cholera, typhoid, plague, tubercle, rabies, and the streptococcic and pneumococcic infections. But in these cases where there is bacterial invasion of an extensive nature, as well as an intoxication with bacterial toxins, the results bear no comparison with those obtained from antitoxic sera in the intoxications. Some, however, are not without a value, and quite good results have been obtained with antistreptococcic sera and with the serum against plague prepared by Lustig and Galleotti in Florence.

The specific serum treatment of disease will undoubtedly achieve more definite success when due account is taken of the necessity for polyvalency; that is to say, when as many and as widely different varieties of the specific micro-organism as possible are used in the immunisation of the animal which is to yield protective serum. The researches of Marmorek on streptococci, the work of Herbert Durham, and an examination which I made of the conditions affecting the protective power of antityphoid sera show, among many other observations, that the protective action of a serum against a particular variety of a given bacterium depends on the degree to which the serum has been rendered "special" for that variety of the bacterium in question; that is to say, upon the use of that variety in the immunisation of the animal which yields the serum.

If you desire to put a muzzle on your dog you obviously choose a muzzle that will fit—one that is "special" for your particular dog. That muzzle will probably fit with fair success a large proportion of other dogs of the same breed, and may be applicable more or less to a considerable number of dogs of other breeds in which the head is somewhat similar in size and conformation. But your sheep-dog's muzzle would not be very serviceable for a terrier, and would be quite inapplicable to a mastiff or Newfoundland. And hence, if you required to muzzle all varieties of dogs, you would provide yourselves with all the varieties of muzzles necessary. And certainly you would not say that muzzles were a failure because a particular example failed to fit a particular dog for which it was not made.

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But in the therapeutic use of immune sera the mistake is sometimes made of expecting a particular monovalent serum, a particular muzzle, not only to suffice for all varieties of dogs, but even for the foxes, wolves, and prairie dogs as well. This error is fortunately though somewhat slowly disappearing.

There is another factor also which requires consideration by clinicians; that is, the destruction of bacteria by the phagocytic ferments.

If you were anxious to get rid of dogs entirely because they bite, it would not be enough to muzzle them and turn them loose, because that leaves them capable of producing other dogs which bite; and if you found that they produced new virulent dogs more rapidly than you could make new muzzles it would naturally occur to you to kill the dogs.

Now bacteria multiply at an enormous pace. They must be muzzled, as we have called it, with the antibodies, since they cannot be killed until the immune body is attached to them, but they *must* then be killed by phagocytic ferment, or they will multiply beyond all chance of muzzling, and the unmuzzled individuals will work destruction.

This is what happens when there is deficiency of complement. Accordingly it follows that a due supply of complement is as essential as specific serum. Unfortunately we have no means at present of supplying phagocytic ferment from without. It follows, therefore, that it is essential to secure leucocytosis in infected animals. This can be done in a variety of ways, such as I mentioned in a previous lecture.

It has been thoroughly established in the case of animals that the experimental production of a proliferation and increase of the phagocytes can bring about recovery where death is otherwise inevitable. It rests with the physician to apply these facts to man.

One other fact of prime importance in the serum treatment of disease has been discovered recently, —the fact that an *excess* of immune serum can work serious and even fatal injury. This is a matter of considerable moment.

Neisser and Wechsberg found that on injecting animals with too much serum they died as certainly as if too little immune serum had been given in the specific treatment of infections. This, on investigation, proved to be the result of a deficiency of complement occasioned by the excess of immune serum, as can be proved by experiments on bacteriolysis *in vitro*. The result is brought about as follows:—The affinity of immune body for complement is *not* increased when the former is united to bacteria; it may perhaps even be diminished. Accordingly, on the injection of a marked excess of immune serum, the complement is seized in greater or less amount by the free immune body which remains, and less or even none can be secured by the immune body which has attached itself to the bacteria. Hence the bacteria are not destroyed owing to deficiency of complement.

I was engaged in working on the question of the complement when Neisser's paper appeared, and have been able to confirm these observations. They afford a further argument in favour of the routine production of artificial leucocyte proliferation in association with the use of anti-bacterial sera. Under such circumstances the danger will be greatly minimised, especially as the clinical tendency is usually to give too little rather than too much of the specific anti-substances.

In the administration of the antitoxin also excess must be avoided, since I have found that the effect of giving large quantities of antitoxin which are not employed in neutralising toxin is to arouse the formation of an anti-antitoxin, and this body can prevent the protective action of the antitoxin in a subsequent infection.

From all these facts it follows that though specific sera have a most valuable protective action, yet the inoculation method alone is truly preventive, and confers active immunity against the infective agents; and thus we see that the great work and works of William Jenner, more than a century ago, laid bare the very root of the whole question of protection from the specific infections, and that the means for its production, found by him for smallpox, was then and is to-day the only safe and certain method of defence against the invasion of the body by bacteria in natural disease.

Application to prevent the spread of Erysipelas.—℞ Argenti nitratis, gr. lxxx. Acidi Nitrici, ♏vj. Aquæ destillatæ, ʒss. M.f. solution. To be painted over the inflamed surface, and for two or three inches beyond it, two or three times a day. (*Higginbottom.*)

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BILHARZIA HÆMATOBIA.*

By J. H. BRYANT, M.D., F.R.C.P.,
Assistant Physician to Guy's Hospital.

GENTLEMEN,—Some cases of hæmaturia, caused by the presence in the body of a parasite, the *Bilharzia hæmatobia*, are the subject of my lecture to-day. I well remember, some time ago, when I was a medical ward clerk, seeing what was supposed to be the first case of this nature at Guy's Hospital, for then it was an affection very rarely seen in this country. The patient was a man who had contracted the disease in Egypt, and after returning to England he was doing the round of the London and Provincial hospitals, and providing the students, for a consideration, with specimens of the ova of the *Bilharzia* from his urine.

Unfortunately it is now less rare, and is likely, when the war in South Africa is over and the soldiers return to their homes, to be by no means an uncommon cause of hæmaturia and other urinary troubles in all parts of Great Britain and her colonies; and it is of great importance for you to be able to recognise such cases when you come across them. At present it is endemic only in Africa and Arabia, but with so many men infected returning to Great Britain, India, Ceylon, Australia, and other parts of the world, it would seem that a grave danger exists of this disease becoming endemic in these countries also, should a suitable intermediate host exist in waters contaminated as they must be by the ova which are passed in thousands with the urine of those affected.

CASE I.—Wm. E. T—, æt. 28, a mechanical engineer, was admitted under my care on April 12th, 1901, for hæmaturia.

He has always been strong and healthy. Twelve years of his life he has spent in the Brazils, enjoying good health all the time. Early in 1899 he

* A clinical lecture delivered at Guy's Hospital, May 7th, 1902.

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went to East Africa to a place on the Zambesi River, not far from Blantyre. From the eighth to sixteenth months of his stay there he had several attacks of malaria, notwithstanding a regular daily dose of quinine. He returned to England in December, 1900, and soon after noticed that on micturition the last few drops which he passed were very dark, and on some occasions distinctly bloody. Two months afterwards he went to see Dr. Crosse to be examined for a new African appointment, and on account of the condition of his urine he was not passed. The blood has been fairly constant since he first noticed it, but not sufficient to colour the bulk of it. There has been no tenesmus. During the last fortnight he has felt a slight, constant, dull pain over his sacrum and the lower part of his buttock. He stated that his food in Africa had consisted of goat's flesh and fowl, vegetables grown in the factory garden, fish from the Zambesi, and watercress grown in a small stream near the works; the water drunk was rain-water collected in tanks. He did not actually bathe in the river, but water from the river was used to supply the house baths.

On admission he was well built and healthy looking. Temperature 98°, pulse 76, respiration 16. He said he felt perfectly well. He was not anæmic. Urine, 1006; no blood present, but a trace of albumen. Microscopically there were many pus-cells and typical ova of the *Bilharzia hæmatobia*. The spines of the ova were terminal. On centrifugalising the urine and treating the deposit with an equal part of warm water the embryos were hatched, and moved about on the slide with great activity. Movement was effected by the protrusion of the anterior part of the embryo, followed by a retraction of the posterior part. The embryos contained ten large refractile bodies and many granules. The circulatory and respiratory systems were normal.

On the 19th he was treated with 5-minim doses of Glycerinum Acidi Carbolici. He went out on the 29th. For several days previous to this neither ova nor blood had been found in the urine. He was advised to continue the treatment.

Case 2.—Alf. W—, æt. 20, was admitted under my care on September 12th, 1901, for hæmaturia and pain in the loins. He enlisted in May, 1896, and was sent out to South Africa on January 4th, 1900; he was in the Royal Engineers, and accompanied Lord Methuen's and Lord Roberts's forces through the Orange Free State and Transvaal. He suffered from two attacks of enteric fever whilst in South Africa. He first noticed blood in his urine after leaving Rustenburg, where he had bathed a good deal in a spruit. In May, 1901, some time after the first onset, he fell down in the street and became unconscious; he was taken to a hospital, and was told that he had run down from loss of blood, and the hæmaturia was put down to the bathing. At this time he was suffering from a good deal of pain in his loins. He had been treated with turpentine internally and hot fomentations to his back. He was invalided home, and whilst in the hospital ship his bladder was washed out with boracic acid and carbolic acid lotions, but with very little, if any, good effect. He says that he passes urine without any blood at first, and that the blood comes with the latter part of it. He occasionally passes clots and what he calls pieces of flesh. The passage of blood has not been constant, but has ceased from time to time for two or three days. Muscular exertion generally brings on or intensifies the hæmaturia. He has also found blood in his motions, and this was the first symptom.

Condition on admission.—He was well developed and looked strong and healthy. The urine was straw-coloured and smoky, sp. gr. 1015, slightly acid, urea 1·5 per cent., small quantity of albumen present. There was very little blood. Microscopically red blood-corpuscles, pus-cells, and ova of the *Bilharzia hæmatobia* were found. The heart, lungs, and abdomen appeared to be normal. He was treated with 10-grain doses of urotropin in cachets. On September 18th he passed blood in his motions, but no ova could be found. Rectal injections of 1 in 5000 formalin to Oiss were ordered every morning. On the 22nd there was again blood in the motions. On October 1st both the urine and the motions contained bright red blood-clots.

He was discharged on October 3rd looking quite well and not complaining of any pain. He said he was passing more blood in the motions than when he came in. The number of ova of the *Bilharzia* in the urine was certainly much less than when he was admitted. He was ordered urotropin to take at home.

On October 29th he was readmitted, as he had

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been passing a great deal of blood both in his urine and in his motions. The week before he was readmitted he suffered from rigors and attacks of fever. I saw him at my out-patients', and found him looking very anæmic and ill. The history of rigors and fever made me suspect that he was also suffering from malaria.

On admission he was anæmic, drowsy, and complaining of severe headache. On October 31st the temperature rose to 102·2°; at six p.m. he had a severe headache, and about 6.30 p.m. he had a rigor, after which the temperature fell. As a careful examination of several stained blood-films failed to show any malaria parasites, a bacteriological examination of the blood was made, and a pure culture of streptococci was the result. He was skiagraphed, but there was no sign of renal or vesical calculus. A few days after, on examining a stained specimen of a centrifugalised deposit of the urine, I found a few chains of streptococci and some cocci. On November 3rd the temperature rose to 100·2°, but he had no rigors after the 1st. The urotropin was omitted, and he was put on benzoate of ammonia and quinine.

On November 4th the temperature rose to 102·2°, and he had a severe rigor; in the evening the temperature fell to 97·6°, and he had another rigor. A careful examination of several blood-films was made, but no malaria parasites were seen. On November 5th he felt better, and the temperature was 100·8°. On November 12th a blood count showed: red blood-corpuscles 4,020,000, white blood-corpuscles 14,000, and hæmoglobin 70 per cent. He had rigors off and on until the 15th, when he felt much better, and rapidly improved, and was discharged on November 26th.

Case 3.—Francis C—, æt. 23, soldier, was admitted under my care on April 19th, 1902, for hæmaturia. He had always enjoyed good health until he went to South Africa, where he had mild attacks of enteric fever and malaria. The malarial attack occurred eighteen months ago. Whilst in the Transvaal on the Crocodile River, his regiment bathed in and drank the unfiltered river water. No other man in his regiment (8th Corps Oxfordshire Company M.I.) developed hæmaturia as far as he knew, but several men in other regiments were affected, the disease in each case being traced to the Crocodile River. He was invalided out of the army for Bilharzia hæmatobia. He was treated with the other men in a South African hospital, but there was no improvement. On returning home he went to the Guards' Hospital and was discharged as uncured. At Woolwich his bladder was washed out, but with very little, if any, improvement. He first noticed blood in his urine in July last. Before this for some days there had been blood in his motions. After active exercise the blood would be much increased in the urine, it being passed almost pure.

Condition on admission.—He is not anæmic. He looks strong and well. The urine is first passed quite clear, and at the end of micturition about a teaspoonful of blood is expelled. Micturition is not painful until the blood flows, when a pricking sensation is experienced along the urethra. The frequency of micturition is not increased. Urine 1015; neutral; albumen present 1 part per 1000; blood present (Guaiacum test). Microscopically many red blood-corpuscles and the ova of *Bilharzia hæmatobia* in large numbers.

A blood examination showed 5,700,000 red blood-corpuscles and 16,562 white blood-corpuscles, a large proportion of which were eosinophiles. On the 21st, in addition to the blood and ova in the urine, pus-corpuscles were also found.

He was first treated with urotropin and Ext. Filicis Maris in capsules. Before going out he was given 4-grain doses of methylene blue three times a day for one day every week, followed by—

℞ Ammon. Benz. ... ... gr. xv
Tinct. Hyoscyami ... ℥xxx
Sp. Chloroformi ... ... ℥x
Inf. Uvæ Ursi ... ... ℥j

three times a day for the rest of the week. He has much benefited by the treatment.

Cases 1 and 3 may be looked upon as typical examples of this disease. Also Case 2 when admitted on the first occasion.

Probably the first mention of this affection is by Chanotin, who in the year 1812 drew attention to the prevalence of an endemic form of hæmaturia in Mauritius. It was left, however, for Bilharz to discover the true pathology of this kind of hæmaturia. In 1851 he was engaged with Griesinger in the investigation of the diseases prevalent in Egypt, and found in the portal veins a trematode worm which he named the *Distoma hæmatobium*. Griesinger in 1854 suggested that this parasite was

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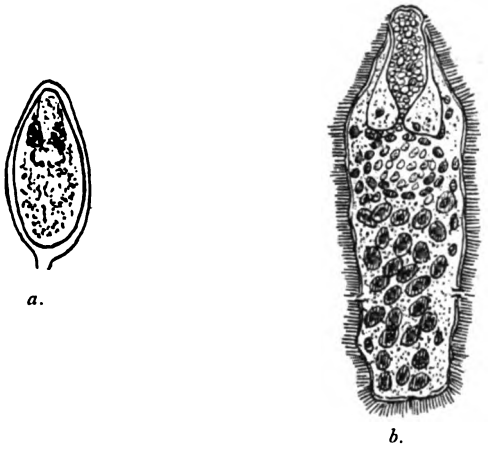
the cause of endemic hæmaturia in other countries. The parasite was subsequently named by Cobbold the *Bilharzia hæmatobia*, the name which has been generally adopted. Dr. Harley recorded the first case in England in 1863, having detected the ova in the urine of a man who had been attacked with hæmaturia in Cape Colony.

Description of the parasite.—The *Bilharzia hæmatobia* is a bisexual trematode worm. Both male and female are provided with two suckers, the foremost of which surrounds the mouth, the other, which is close to it, being used as an organ of fixation.

The male is milk or opal-white in appearance, and measures about half an inch in length (7 to 16 mm.). Its upper surface is convex and covered with tiny ciliated tubercles; the sides of the body



a. *b*.

a, Ovum with terminal spine; *b*, Embryo.

are flattened and bent so as to overlap and form a central gynæcophoric canal, in which a part of the female lies during coition.

The female is darker, more cylindrical, and longer than the male, measuring about three quarters of an inch in length, and looking like a silk thread. The body is smooth and not covered with cilia. The female is usually found lying in the gynæcophoric canal of the male with her extremities free and projecting. Both have a digestive apparatus which ends in a blind intestine which is often filled with blood-corpuscles, showing that the worm lives on the blood of its host.

The adult worms are usually found in the portal vein and its tributaries and radicles. They are also found in the veins of the bladder, uterus, ureter, rectum, and rarely in the inferior vena cava. As many as three hundred have been found in the portal vein and its branches in one case. Davidson writes, "To insure finding the worms in the portal vein it is as well, as I suggested so far back as 1875, to tie the portal vein with a double thread before cutting it so as to prevent the escape of the blood." Ova and egg-shells have been found in the heart. The ova can be seen in the uterus of the female if examined under the microscope. They are oval, resembling a melon-seed in appearance, and measure from $\frac{1}{200}$ in. to $\frac{1}{100}$ in., usually $\frac{1}{170}$ in. in length. One end is blunt and the other is furnished with a sharp projecting spine. Ova are sometimes found with the spine placed laterally. Zancarol states that the spines are terminal when the parasite inhabits the veins of the urinary tract, and that lateral spines are found when it occupies the veins of the intestine. The particular significance of the position of the spines does not appear to be obvious; it has been suggested that it is due to the pressure excited by the muscular coat of the intestine on the ova whilst they are passing from the parent worm. The egg-shell is transparent, so that the embryo can be seen within it, its head being usually placed towards the spine. The embryo is usually matured and ready to leave the egg. They do not hatch in the urine, but will do so readily in water at 80° F. The egg-shell bursts in a longitudinal direction and the embryo escapes.

Ova may be found in various parts of the body, the favourite situations being the walls of the bladder and rectum. They have also been found in the ureter, lungs, skin, heart, and but rarely, if ever, in the stomach, pancreas, or spleen.

There appears to be substantial evidence that ova are hatched while in the body, for empty egg-shells have been found in various organs. Lieut. Lelean, at a meeting of the Medical Society of London, demonstrated hatching of the ova in blood-serum outside the body. The embryo is ovoid whilst still in the egg, but as soon as it is hatched it becomes more elongated. It has an oval proboscis, which is continually moving forwards and backwards. All the body except the proboscis is covered with cilia. The body wall contains longitudinal and transverse muscular fibres. It is furnished with a primitive digestive canal, nervous and water vascular system, and large germinal cells, which probably develop into

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rediæ. It moves about with great speed, and alters into all kinds of shapes.

Life-history.—The complete life-history has not been worked out, and can at present only be conjectured from analogy. Sonsine a few years ago believed that he had discovered the intermediate host, but subsequently abandoned his view. The life-history is probably very similar to that of the liver-fluke. Ova are passed with the urine and fæces direct into ponds and streams, or are washed into them by rains; the embryo hatches and penetrates the body of some fresh-water mollusc, forms a sporocyst which contains rediæ, inside of which are developed the cercariæ, which are larvæ with tails resembling tadpoles. The cercariæ are swallowed most probably in water and enter the body in this manner.

Distribution.—*Bilharzia* has been found in many parts of Africa. It is most abundant in Egypt. Its presence has been ascertained in many parts of South Africa, especially in the eastern part of Cape Colony, at Uitenhage, Port Elizabeth, Grahamstown, Fort Beaufort, Alice, King William's Town; in certain parts of Natal, around Durban and Pietermaritzburg in particular; in the Transvaal beyond the Magaliesburg, especially in the Rustenburg district and on the Crocodile River; further north, between Zanzibar and the Zambesi; in the Nyassa district; in the Hinterland of Tripoli and Algeria; in the countries round Lake Chad; at Accra, on the Gold Coast, and other places on the West Coast of Africa; in Madagascar, Mauritius, and Réunion; in Arabia; and cases have also been reported in Sicily, Cyprus, and India. From this very wide distribution it will be seen that the possibility of other countries being infected is not at all improbable, and it appears to me that the chief danger lies in the direction of India and Australia. Apropos of this possibility, Davidson writes, "Only lately (1888) the occurrence of two cases in England referred to by the late Berkley Hill leads us to suspect that Bilharzia may be contracted even in countries with a temperate climate such as England."

The mode of infection is not known for certain, but those who have had an opportunity of studying the disease where it is endemic are almost unanimous in believing that it enters the human organism by means of drinking water, or with uncooked vegetables, in the form of a free larva or a larva within the body of its small intermediate host. Guillemard appears to favour the view that the armed embryo enters the body through the skin, urethra, or anus during bathing, and he writes as follows:—"It is not even certain that it occurs by the ingestion of the animal. Many competent authorities believe that the armed embryo directly penetrates the skin, urethra, or anus during bathing. In no other way is it easy to account for the absence of the disease in infants and young children, and for the very remarkable fact that among European females it is extraordinarily rare. Thus Brock in South Africa, among many hundreds of cases, never saw a female affected. River-bathing is very seldom indulged in by women in South Africa, but the male youth are much in the water. Among natives the women are apparently more frequently the sufferers, but they have often to ford streams, and may daily be seen wading up to their knees to fill their jars and calabashes for household use." These appear to be strong arguments in favour of this method of infection. It is also evident that the natives themselves believe they are infected in this way, for when they bathe they attempt to protect themselves by tying a string around their foreskins. I am informed that our soldiers emulated the natives in this respect, but without result, as in spite of this precaution they became infected. The three patients who have been under my care all attributed this disease to bathing. The patient from the Zambesi district took his morning bath in water obtained from that river, and drank water which had been collected in tanks; but this is no proof that he contracted the disease in this manner, for he distinctly stated that he had eaten watercress grown in small streams near the works. The two other patients both were affected when in the Rustenburg district, a part in which the disease is most prevalent. Col. Gubbins, R.A.M.C., speaking at the Medical Society of London a week ago, stated that nearly all the cases he had seen in South Africa came from this part of the country. Both men had bathed, but had also drunk water from the same source. The man who has just left John Ward stated that one day when bathing he was helping a comrade to swim, and was pushed under the water and swallowed a good deal. Davidson considers that the embryo gains access to the body by means of drinking water, and remarks that the other view is most unlikely, as there is no known example of an entozoon intro-

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ducing itself into the human body in this manner; and a further point against this supposition is the fact that many people have been affected without ever having bathed—a remark which particularly applies to cases in Egypt. Another point of great interest in connection with the mode of infection is the fact that in a very large number of the cases the first symptom of the disease is a pseudo-dysentery characterised by diarrhœa and the passage of blood. This would suggest that the parasite invades the body by means of the rectum, and from the hæmorrhoidal veins and plexus gets into the tributaries of the portal vein and into the vesical veins.

To sum up, however, the evidence in favour of infection by means of drinking water appears to be most conclusive. That bathing is an important factor I have no doubt, but I do not believe the embryo gets into the body by means of the urethra or the rectum, but by water swallowed or taken into the mouth.

The period of incubation appears to vary; it may be as little as four months. In Natal many of the coolies show signs of the affection within six months of their arrival in the country. The disease in South Africa is most frequent in young boys, but is very rare before the age of five or six. A correspondent in the 'Brit. Med. Journ.,' p. 1000, vol. i, 1900, writes that the disease appears to be spreading in South Africa, and that in many of the native schools in East Griqualand nearly all the children are affected.

The symptoms vary very much in degree. In some cases the indications of the disease are so slight that the patient experiences very little or no trouble at all. He may simply notice that he passes a little blood at the end of micturition. Case 1 is an example of such a mild infection, for the patient was not aware that there was much the matter with him until he was examined by Dr. Crosse, for he thought that his urinary trouble was probably a sequela of malaria. Hæmaturia is by far the most common and characteristic symptom. The amount of blood varies very much, from almost pure blood to a mere trace at the end of micturition. One of the earliest symptoms in a large number of the cases is a sudden attack of diarrhœa accompanied by the passage of blood and mucus, or by the passage of blood, mucus, and scybala, so that in the early stages of the disease the patient may be thought to be suffering from dysentery. Micturition may be painful, especially the end of the act when blood-clot and *débris* may be passed. There may be pains in the loins and perineum, in the thighs, inguinal and hypogastric regions. The amount of blood in the urine and the intensity of the pain may be much increased by active exercise, especially horse-riding, so that if at all severely affected the sufferer becomes incapacitated as a soldier.

If the prostate and vesiculæ seminales are affected there may be severe perineal pain, priapism, and seminal emissions. If the neck of the bladder is involved there may be vesical tenesmus. If the patient loses much blood and suffers a good deal of pain, he complains of lassitude, becomes anæmic and weak, and may suffer from epistaxis and fibrillary muscular tremors.

It is so common in Natal that it is looked upon as being almost physiological, and boys appear to take very little notice of the complaint. Guillemard states that it is a noteworthy fact that the shirts of patients affected with Bilharzia are almost always stained with blood. The hæmaturia may last for years, and then may begin to subside, and in some cases disappears altogether.

The *urine* shows characteristic changes. At first it may be quite clear, and on close examination may show the presence of a few minute specks of a brilliant scarlet colour. Later, if the act of micturition is watched, the first part passed may be quite clear, but at the end of the act a drachm or less of blood and filaments of mucus may be ejected. In some cases, especially after muscular exertion, almost pure blood may be voided.

The amount is not altered; the colour is generally darker than normal, varying, however, according to the amount of blood. It may have a sweetish or ammoniacal odour. The reaction may be faintly acid or alkaline. It may be turbid, and long colourless filaments like threads of fine white silk, and containing minute yellowish-white specks, may be seen.

There may be a deposit of blood-stained, branching, irregular, shaggy, vein-like masses, which one patient compared to lumps of flesh. Flat blood-clots may also be present. Calculi may form, composed of nuclei of aggregations of ova covered with incrustations of phosphates. In Egypt patients are sometimes seen with very large vesical calculi.

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Microscopically red blood-corpuscles, pus-cells, epithelial cells, ova with terminal spines single or in masses, and oxalate of lime, uric acid, or triple phosphate crystals may be seen. In some cases thousands of ova may be passed in a day.

The most important complications are cystitis, urinary calculi, new growths in the bladder or rectum, occlusion of the ureter leading to hydronephrosis, pyelitis and pyelo-nephritis, hepatic abscess, localised peritonitis, and perforation of the bladder.

The vesiculæ seminales may become involved, causing spermatorrhœa, and the ova may be found in the semen. The rectum may be involved and much blood and mucus be passed, accompanied with severe tenesmus. The ova are rarely found in the stools.

Morbid Anatomy and Pathology.—The most important change is chronic cystitis. There is swelling, hyperæmia, and ecchymosis of the mucous membrane of the bladder, with thickened patches and papular or warty-looking elevations covered with thick mucus or incrustations of urinary salts. The bladder may contain calculi, and its muscular coat is usually much thickened. The ureter and the pelvis of the kidney may also be affected, but in the latter part it is rare to find any changes. When the ureters are affected, obstruction may occur with hydronephrosis as a result. The mucous membrane of the lower part of the rectum may be thickened, and in this position there may be polypoid growths, which may be mistaken for hæmorrhoids. Ova and empty shells may be found in the diseased parts and in the mucus on the surface of the bladder. The parasites themselves are found in the portal vein and its radicles and branches. Ova have also been found in the heart, liver, kidneys, and even in gall-stones. In the bladder the ova are mostly situated in the submucous coat, where they occur in groups, surrounded by a thin capsule of connective tissue.

It is not quite clear how the ova get into the mucous membrane of the bladder and rectum. "It is possible that the sharp spine with which each of them is provided enables them to work their way gradually outwards under the influence of pressure to which they are subjected by the contraction of the muscular wall of the bladder." Cobbold has suggested that the spine acts as a kind of "holdfast" to aid the violent efforts of the embryo to get out of its shell. The hæmorrhage is caused by the ova blocking small blood-vessels and leading to blood extravasations, local inflammatory changes, and hyperplasia of the mucous membrane. The intimate relation which exists between the systemic and the chylopoietic veins accounts for the frequency with which the bladder and rectum are affected.

Diagnosis.—The diagnosis does not as a rule present much difficulty. The curious flesh-like masses in the urine are characteristic. The urinary sediment must be carefully examined under the microscope, and if ova with terminal spines are found the diagnosis is obvious. It is best to catch in a watch-glass the last two or three drops of urine which the patient passes, and to prepare from this the specimen for microscopical examination.

Ova are rarely found in the fæces. If the symptoms are mainly intestinal in character, blood and mucus being passed *per rectum*, dysentery may be suspected, and many cases of Bilharzia hæmatobia which commence in this manner are diagnosed as such. If the slightest amount of blood is present in the urine in such a case, Bilharzia hæmatobia should be suspected and the urine examined for ova; in fact, it would be a wise precaution to make a point of centrifugalising the urine of every patient from Africa who suffers from symptoms of dysentery, and examining the deposit in order to determine or exclude the presence of these characteristic spine-tipped ova.

Prognosis.—Boys often appear to be cured at puberty. Adults are rarely quite sound again after once being infected, for the life of the parasite corresponds with the life of its host. Guillemard mentions a case in which an acute attack of hæmaturia occurred eighteen years after the onset of the disease. The greater the number of parasites present in the veins the more severe is the disease and the worse the prognosis. Although the hæmorrhage may be severe at times and produce severe anæmia, it is rarely the immediate cause of death; pneumonia or cystitis and its complications are the commonest causes.

Treatment.—As it appears that the *Bilharzia hæmatobia* embryo enters the human body after having gained access to an intermediate host which inhabits fresh water, the following preventative measures should be taken in an infected district:

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1. As far as possible the ova should be prevented from getting into fresh water by efficient drainage, and the inhabitants should be cautioned about micturating into or near a river, lake, pond, or other source of fresh water.

2. Drinking-water should be boiled or filtered, and children should be particularly warned against drinking water from ponds, streams, or canals.

3. No uncooked vegetable food, such as watercress, which has grown in an infected district, should be used as food.

4. Bathers should be cautioned to be careful to avoid drinking, or even taking any water into their mouths, whilst bathing.

Rest is essential, for any excessive or violent muscular exertion increases or brings on the hæmorrhage. Riding, in particular, must be discontinued, as it increases the hæmaturia. Chills must be carefully guarded against, and a flannel belt should be worn round the loins. The diet should be plain and simple, and stimulants should be avoided.

There is no drug which is known to effect a radical cure by exterminating the parasite or eradicating it from the system.

Urotropin has been given an extended trial, but has had no appreciable effect except as a urinary antiseptic in cases complicated by cystitis.

Male fern has been tried, but with no beneficial result. Lieut. Lelean, R.A.M.C., recommends methylene blue as the most useful drug known, and my small experience corroborates this view. It is best to give four-grain doses three times a day for one day every week, and on the six other days to give a mixture containing ten- or fifteen-grain doses of benzoate of ammonia. The methylene blue has a remarkable effect in checking the hæmaturia, and patients experience great relief from its administration. The benzoate of ammonia lessens the irritability of the bladder.

Dr. T. S. Dowse says, in 'The Pocket Therapist,' that he has seen the following draught act with marvellous effect in many cases of delirium tremens :— ℞ Liq. Opii Sedat. (Battley) ♏xl, Liq. Cinch. (Battley) ♏xxx, Aquæ ad ʒij. M. ft. Haust. To be given with 6 oz. of brandy beaten up with two eggs.

LECTURES ON SOME AFFECTIONS OF THE HIP.

Lecture II.

COXA VARA.

A Surgical Demonstration at the Medical Graduates' College and Polyclinic.

By A. H. TUBBY, M.S., F.R.C.S.,

Surgeon to the Westminster Hospital; Senior Surgeon, Evelina Hospital for Sick Children, and Consulting Surgeon to the Hip Hospital, Sevenoaks.

Gentlemen,—The subject which I have chosen to-day is coxa vara, and it is one which has recently come into notice, although the deformity has been recognised for some considerable time. The reason being that the deformity is situated in the hip-joint, and it has often been mistaken for incipient tubercular disease or for congenital displacement of the hip. But now that its existence can be verified by the Röntgen rays, there is no excuse for overlooking or mistaking it. As the condition is somewhat novel to many, and has not hitherto found its way into the general text-books, I have taken the opportunity of making a written abstract of the subject and of laying it before you. It seemed to me well that you should have the condition clearly sketched out, so that you might be able to follow not only the subject of coxa vara, but its diagnosis from other conditions.

Firstly, with regard to the definition. Coxa vara is a depression of the head and neck of the femur resulting in diminution of the angle between the head and neck and the shaft. This (Fig. 1) represents the normal inclination of the head and neck of the femur to the shaft. The deformity of coxa vara consists in first of all a downward sinking of the head of the femur, together with a bending of the neck, and the result is that you find the appearances in the illustrations 2 and 3; so that the line between the top of the trochanter and the uppermost part of the head of the femur is no longer oblique, but becomes almost horizontal. That is the condition which obtains in a case of medium severity (Fig. 2). But in a case of great severity the relations of the trochanter to the head of the femur are totally altered, because the head of the femur may, even at its highest part, be a considerable distance below the top of the trochanter, and there is also

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some absorption of the neck. The nature of the deformity and the way in which it comes about explain many of the symptoms which are met with, but some are a little difficult to follow, because the deformity is not so simple as I have represented here. Not only is there a bending down of the neck of the femur, but it may be twisted either backwards or forwards, so that it becomes convex backwards or convex forwards. So there are two causative factors of symptoms to be considered—first of all the bending downwards of the neck of the femur and sinking of the head, and then the antero-posterior deflection of the neck, which becomes convex either posteriorly or anteriorly. If the head of the femur

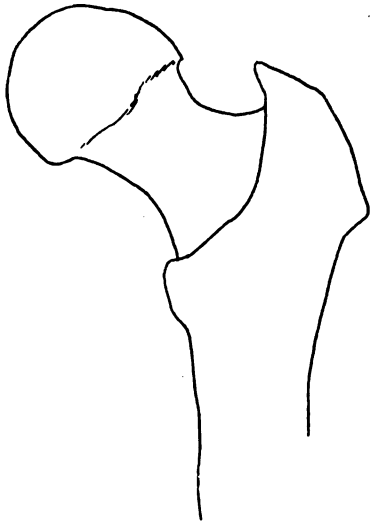

FIG. 1.—Outline of upper part of normal femur.

remain in the acetabulum, as it does in these cases, it stands to reason that there must be some alteration of the inclination of the shaft to the femur; and you will find that as the head sinks, so the shaft of the femur tends to acquire a new axis, and the direction of the axis is more towards the middle line than is natural,—that is to say, the lower end of the shaft of the femur goes inwards. This leads to other symptoms, of which I shall have to speak presently. There is no doubt whatever in my mind that a large number of cases of genu valgum are associated with varying degrees of coxa vara, and to me it is a moot point as to which is the original deformity—whether genu valgum is the cause of coxa vara, or whether the reverse occurs. I think there is a certain amount of evidence to show that in many cases the primary deformity is the sinking of the neck of the femur, then the shaft of the femur inclines inwards, aided also by the pull of the adductors, and thus we have genu valgum, which is so common. As in the case of all newly described affections, many conditions have been designated as coxa vara which really are not so. Coxa vara is a term which should be used for the resulting deformity caused by a bending of the neck of the femur due to softening. Some limit the term to those cases due to rachitic softening; others include all causes, such as tubercular epiphysitis, osteomyelitic softening, and the displacement produced by fracture.

Now with regard to its *etiology* and *causation*. Firstly, coxa vara is eminently a disease or deformity of growing life—that is to say, it is much more common between the ages of ten and twenty than at other times,—and it is also more common in boys than it is in girls. Those facts are now well established by a long series of statistics. I show you now an X-ray print of a case of coxa vara which was associated with very bad genu valgum, and I think you will be able to get some idea of the nature of the deformity from the skiagram. Curiously enough, coxa vara is more often unilateral than bilateral. The unilateral cases are about four or five times as common as the bilateral ones. The most common cause of coxa vara is undoubtedly rickets, the so-called rachitis adolescentium, styled by some people late or recrudescent rickets. This leads me to speak of a point in rickets on which I wish to insist rather strongly. The extensive experience I have had of rickets leads me to believe that it is not only a disease of infancy, or congenital disease, or one of adolescence, such as the term adolescent rickets seems to imply; but I believe it to be a continuous disorder, often commencing *in utero* or in infancy, and in many cases going on to the twenty-second year, with occasional outbreaks and disastrous consequences. In infancy and childhood one sees the most common forms—namely, visceral disturbances and softening of bones resulting in the so-called bandy legs. For a time the child seems to recover. But about the sixth year one often notices oncoming scoliosis, and this may occur as the result of rickets any time from the sixth to the twelfth year. You will see flat-foot beginning at about the twelfth year, and

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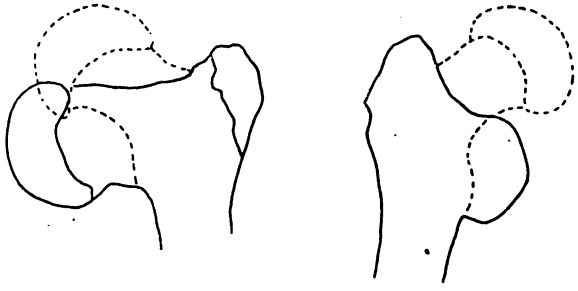
deformities like coxa vara coming on from the tenth to the twentieth year. So I think there is strong evidence to show that rickets is a continuously progressive disease, manifesting itself from time to time in various deformities and troubles. I feel it necessary to insist upon this; otherwise it is difficult to explain these anomalous cases of softening of bone which have been hitherto classed under the term osteomalacia, osteoporosis, and so on. As far as we can ascertain, the most common cause of coxa vara is the so-called rachitis adolescentium, but which I should call "continued rickets." In this disorder there is a softening taking place at the epiphysial line of the neck of the femur, precisely analogous to the softening which you find at the other epiphyses; and the weight of the body pressing on the neck of the femur causes it to bend downwards, and the upper part of the bone to assume an abnormal shape. Among the other causes, which are less common, is the so-called osteomalacia. But I think that is open to doubt. It is very difficult to establish the existence of osteomalacia in children unless they have other typical symptoms. Any cause, such as tubercular disease, which gives rise to inflammation of the neck of the femur, will also necessarily result in its softening and bending. A tubercular focus forms, the bone is softened, and the head sinks down, so that there is a lessening in the angle of the neck and the shaft. This variety has been called tubercular coxa vara. If there be an injury, such as separation of the epiphysis of the head of the femur, which is by no means uncommon, the condition may arise. Some years ago, when I wrote on these epiphysial troubles, and before X-rays were known, I went through the literature of the subject to see how many cases of separation of the head of the femur I could collect. I could only find one case which was verified. But the use of X-rays has shown that there is a moderate number of cases in which traumatic separation of the head of the femur has occurred. If the separated epiphyses should unite badly there is caused an alteration in the angle of the neck of the femur, and hence the term "traumatic coxa vara." There is another kind of coxa vara, in which the bending of femur is just below the trochanters and results in an external convexity at this point. This girl here has the two kinds, as is shown in the photograph. And the first case of bending of the shaft of the femur was described by Mr. Keetley many years ago. The result of such a state of affairs is that there are adduction of the condyles and many of the symptoms of ordinary coxa vara. This last-mentioned form I am inclined to call "false coxa vara," and when the neck alone is bent "true coxa vara."



FIGS. 2 & 3.—Outline of upper third of femur in two cases of coxa vara. (After Whitman.)

Now we shall note how the symptoms arise. Remembering the mode of onset of the lesion, it is fairly easy to grasp them. Let us take this line *a* to be the middle line of the body. Let *e* represent the acetabulum If the neck (Fig. 4, *b*) sink and the angle (*f*) be diminished, the adductor muscles must gain an advantage by their pull. And then what happens is that the lower part of the shaft of the femur is drawn inwards, and there is loss of the power of abduction. This is one of the most prominent symptoms of coxa vara. The onset is determined by a fall, or by strain, or by over-walking or long standing. Some of the cases written of in America have been distinctly traced to boys of weakly physique entering for situations as errand-boys, or other posts requiring much walking and long

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standing. In one case the onset of deformity was due to a boy undergoing excessive fatigue in walking. It is a curious, but a certain fact, that some of the diagnostic signs are most evident when you flex the thighs upon the abdomen. The first point which strikes you about the patient is that there is a limping and a shortening of the limb. But the shortening is not very great as a rule, not more than one half to one and a half inches. Yet some cases have been described in which the shortening is as much as two inches, but such must be exceptional. Here is a child the subject of coxa vara. He has the big stomach of rickets, Harrison's sulcus, the prominent

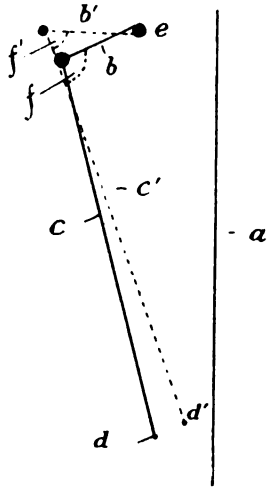

FIG. 4.—*a*, Mid-line of the body. *b*, Normal angle of inclination of the neck of the femur. *b'*, Angle of neck in coxa vara. *c*, Normal axis of shaft of femur. *c'*, Altered axis in coxa vara. *d*, Lower end of normal femur. *d'*, Lower end of femur in coxa vara. *e*, Acetabulum.

umbilicus, and prominent trochanters, as well as genu valgum. His deformity is mainly on the right side; the trochanter is unduly prominent, and there is a considerable tendency to adduction. The X-ray picture shows that the boy has an early degree of coxa vara. If the neck of the femur be convex anteriorly or posteriorly, you will have inversion or eversion of the foot, just as in dislocation of the hip, dependent on whether the head is on the dorsum ilii or on the pubes. There will also be in coxa vara a tilting of the pelvis, and lordosis, and waddling gait, as you see here. If the affection be bilateral there is adduction well marked on both sides, so that the legs tend to cross one another. In this affection you find one cause of scissor-leg deformity and scissor-legged progression. The scissor-legged tendency is not common, but it is seen best when you put the patient on his back and flex the thighs.

One point I must mention is that if you have a case in which the neck of the femur is very convex anteriorly, you will find that the angle of flexion of the thigh is limited considerably, so that there may be a good deal of difficulty in diagnosing that condition from one of hip-joint disease, in which the loss of flexion is one of the prominent signs.

Now with regard to *diagnosis*. First you have to distinguish the condition from hip disease. In both hip-joint disease and coxa vara there is pain at the onset. But the pain of hip-joint disease is persistent, and it is associated with a certain amount of night-starting, with loss of general health, and constitutional symptoms. In both diseases there is shortening. In the first stage of hip disease, as I told you last time, there is apparent lengthening, and you only obtain real shortening when the head is absorbed or has been dislocated. But there is shortening in coxa vara from the first. In hip disease you have adduction too in the late stage, but in hip-joint disease there is limitation of movement in all directions, not only in flexion but in rotation, and also in inversion and eversion; whereas in coxa vara the most striking thing is that the chief limitation of movement is in abduction and slightly in flexion. There are no constitutional symptoms in coxa vara except those of rickets in some cases. But the best means of arriving at a diagnosis is certainly by the use of the X-ray screen. So, then, the cardinal signs of coxa vara are slight pain, shortening, limping, prominence of the trochanter, which is not elevated above Nelaton's line, and inclination of the pelvis towards the affected side, with loss of abduction. The affection is often also mistaken for congenital dislocation, and there is no doubt a large number of cases of congenital dislocation were put down as coxa vara when the latter condition was first talked about. In many cases it is difficult to make a diagnosis between unilateral congenital dislocation and unilateral coxa vara, and I show you the skiagraph of a case in which it was so. In congenital dislocation you have, as a rule, these leading signs. The affection has always been noticed from the

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time the child began to walk. If the case be a fairly early one—that is to say, if the child be under the age of six,—there is no limitation of movement whatever; the movements are perfectly free and good and painless; in fact, they are too free. In a case of congenital dislocation, too, you will note that the head is out of place, and instead of being felt behind the femoral artery it is found up the dorsum ilii behind. Another sign of value is that in congenital dislocation, if you firmly fix the pelvis and take hold of the limb on the affected side, you can push the head up and then draw it down, and this is known as "telescoping." In both affections there are shortening of the limb and prominence of the trochanter.

With regard to the *treatment* of coxa vara in the early stages, that is to say where the shortening is not more than half an inch, and if shortening is going on, the child should be placed in bed with

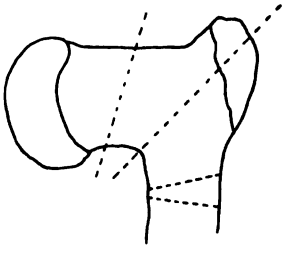

FIG. 5.—To illustrate lines of osteotomy of femur in coxa vara.

the limb in full abduction, and weight-extension put on, and an effort made by these means to increase the angle which the head and the neck of the femur form with the shaft. A considerable number of cases are benefited in that way. I had a young gentleman under my care, aged seventeen, who had half an inch of shortening. I kept him at rest for five or six months, and at the end of that time the half-inch shortening was completely gone, and he walked as well as before. The difficulty is to be able to tell when you should allow the patient to get up again. This was the question which I was asked:—How shall we know when we may let him get up again, and when to stop the extension? I said, "If you find the extension is producing no more lengthening, and you let him continue in bed for two months after that, you may conclude that the softening process has ceased, and it will then be safe for him to get up. When you cannot draw down the limb any more it shows that the bones have become firm again."

Now let us look at this child which has been brought to us. She has limitation of abduction in the left leg. There is very little difference in the measurement of the two legs from the umbilicus. There is no telescoping. I think it is an early case of coxa vara. The main diagnostic sign is the absence of abduction, one of the earliest signs which you see. Such a case as this would be best put to bed and weight-extension put on the leg, as much in a position of abduction as possible. But in more severe cases you have to resort to other measures. Suppose you have a case of this description (Fig. 5), and it is impossible to restore it by the method of extension or by recumbency. You may either divide the neck of the femur, or the trochanter obliquely, or the shaft of the femur below the trochanter. Treatment by means of an oblique section through the trochanter or a transverse section below the trochanter has been much discussed. At present the fashion seems to be to divide below the trochanter, remove a wedge of bone, and bring the lower fragment well outwards to a position of abduction. I prefer always to divide obliquely through the trochanter after section of the adductor tendons, so as bring the limb out, and for these reasons: that you have a bigger surface for bony union, you open the angle so as to leave a wedge-shaped space below which can be filled up by good sound bone, and you are not then troubled with any external deformity, as you might be if you divided immediately below the trochanter. In very severe cases it may be necessary to remove a wedge-shaped piece from the shaft of the femur and so bring the lower part of it out, and thus obtain the required abduction. When shortening is once firmly established you can compensate it by means of a high boot.

Lateral Section of the Pelvis.—Two cases of lateral section of the pelvis as suggested by Gigli, with perfect results for both mother and child, are reported by Pestalozza ('Zeit. für Gyn.,' No. 4). Comparing these with seven symphyseotomies, he favours the former procedure as giving practically no hæmorrhage. — *American Journal of Obstetrics*, May, 1903.

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A DEMONSTRATION OF CLINICAL CASES.

Given at Charing Cross Hospital,

By HERBERT F. WATERHOUSE, F.R.C.S.,

Senior Assistant Surgeon, Charing Cross Hospital.

GENTLEMEN,—I wish to-day to show you some eight cases of more or less surgical interest.

CASE 1.—NEURALGIA OF THE INFERIOR DENTAL NERVE OF GREAT SEVERITY TREATED BY NEURECTOMY.

This patient presented an example of very aggravated neuralgia of the inferior dental nerve. It is very nearly three years ago since this man first came under my care with most agonising neuralgia of the dental nerve of the right side,—that is, neuralgia of one single branch of the trigeminal nerve. My experience of these cases of intense neuralgia treated by operative measures has hitherto, I think I may say, been somewhat disappointing. In a large number of cases of intense trigeminal neuralgia, if you divide or remove one nerve you find the pain recur in a neighbouring branch. But this man's case is satisfactory in that the pain, which was so agonising as to make him weary of life, has been greatly relieved, with very slight cosmetic ill-effect on the face. I performed the following operation:

I made an incision along the posterior part of the lower margin of the jaw, and curving upwards for half an inch behind the angle of the jaw. I then separated the insertion of the masseter with scissors and periosteum elevator, and retracted the whole of the soft parts upwards so as to expose the ramus of the jaw. Then with a small trephine I removed one third of an inch of bone from the centre of the ascending ramus, and found at once the inferior dental nerve, which I divided, and, making firm traction on the distal end, I pulled out of the bony canal in which it lies about one and a half inches of it. From that moment the pain ceased entirely, and the patient, who for six months had lived in misery, enjoyed two and a half years of complete freedom from pain. At the end of that time a slight pain returned in the distribution of the nerve, one and a half inches of which I had cut away. The pain—never, as the patient tells us, really severe—persisted for some time, and I gave him tincture of gelsemium in doses of fifteen minims, and butyl-chloral hydrate ten minims, three and sometimes four times a day. All pain has now disappeared. I wished to show you the case because it is an encouraging one. By operative treatment we entirely removed for two and a half years the terrible agony suffered by the patient, and although the pain returned for a few weeks it proved readily amenable to treatment by drugs. Prior to operation I had tried treatment by drugs for a long time without any effect whatever. I should, moreover, like to draw your attention to two points. After the operation the side of the face was paralysed for four or five days, owing to the fact that in retracting upwards the soft parts superficial to the bone I had stretched the facial nerve. This condition, however, passed off completely in a short time. Healing by first intention occurred, and, as you will see, there is no interference with the movements of the jaw, and the scar hidden by the beard is not noticeable.

CASE 2.—PARENCHYMATOUS GOITRE TREATED WITH IODINE ADMINISTERED INTERNALLY, AND APPLIED LOCALLY TO THE SKIN COVERING THE GOITRE.

I have here two cases of goitre to show you. In the first place notice this child's neck. This girl, Lillie K—, aged eleven and a half, presents an example of parenchymatous goitre. She first came under my care on the 14th April last with a large and very hard parenchymatous goitre. The thyroid enlargement was fairly symmetrical. Many people mistake these cases of parenchymatous goitre tensely filled with secretion for fibrous goitre. In fact, this patient was sent to me as a good example of very hard fibrous goitre. As a matter of fact fibrous goitre is a condition that I have scarcely ever seen. Almost every case I have examined that was supposed to be fibrous goitre has turned out to be parenchymatous goitre, with the vesicles so tensely filled with thyroid secretion that the goitre felt almost as hard as stone. As far as I am concerned fibrous goitre practically does not exist. I am certain that almost all these cases of stony-hard regular enlargement of the gland are simply instances of parenchymatous

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goitre. I need not remind you how often a very tense cyst feels like a solid tumour. I show you this case because its treatment has been extremely satisfactory. In many text-books you will read that the treatment of goitre by medicinal means is highly unsatisfactory. As a matter of fact the treatment by medicine of parenchymatous goitre, with more or less general enlargement of the gland, is, in the majority of cases, I think, certainly distinctly satisfactory in diminishing the size of the gland, and in relieving the symptoms from which the patient suffers. This little girl came with a goitre ten weeks ago, I am sure three times as big as it is now.

What has been done for it? I may say that in the large majority of cases of parenchymatous goitre immediate surgical treatment is quite unjustifiable. It is certainly unjustifiable to operate for parenchymatous goitre without preliminary treatment by drugs unless great dyspnœa is present, and great dyspnœa is unusual in this affection. The reason is that the trachea, I take it, is not pushed to one side; it remains in the middle line, because there is a swelling of about equal size on either side. Unless dyspnœa is a marked symptom, then, I do not think that operation in the case of parenchymatous goitre is justifiable. Certainly I do not think it justifiable simply to rid the patient of what is only an unsightly and not a dangerous tumour. How has this girl been treated? She has been treated in the way in which you will get a very considerable amount of success in two out of every three cases of parenchymatous goitre. There are only two remedies that are, in my opinion, of the slightest value in the treatment of parenchymatous goitre—iodine and thyroid extract,—and of the two iodine is infinitely the more valuable. Thyroid extract, in certain cases, has given me very good results; in others it has certainly aggravated the condition, and sometimes brought on symptoms of exophthalmic goitre. The treatment adopted in the case of this girl has been that she has had two minims of the tincture of iodine in a tumbler of water three times a day after food, and she has had the skin over the goitre freely rubbed with an ointment containing eight grains of biniodide of mercury to the ounce of lanolin. That is the sole treatment adopted in this case, and the goitre has all but disappeared. You can see that she has still an enlarged thyroid, but what was previously a "woodenly" hard, tense, swollen goitre has now become quite soft, and has diminished in size until now it is only one third of its previous bulk. The whole of this improvement has taken place since the 14th April. This is by no means a very exceptional case, though the beneficial effect of the iodine is usually less rapidly obtained. In the majority of cases of parenchymatous goitre that I have treated the improvement has been, though not as rapid as in this instance, certainly very satisfactory. One of the most markedly beneficial results that I have seen was that of an old gentleman seen in consultation with my friend Dr. E. Seton Pattison, of Fulham, in whose case the circumference of the neck was reduced two and a half inches in eight days by the action of iodine, with the result that the symptoms of pressure, previously markedly present, entirely disappeared. This gentleman has still a goitre, but it now gives him no trouble at all. In the case of this girl I should like you to feel the thyroid, as some of you saw and felt it ten weeks ago. I may say that the ointment makes the skin desquamate a little at first, but that very soon passes off. The gland was, as some of these tensely filled glands are, somewhat painful to touch, but this symptom disappeared within a few days of the commencement of the treatment. Of course, what the iodine does is in some way or other to promote the absorption of the colloid material, but the exact way in which this is brought about is not clear.

Case 3.—An Adenomatous Tumour partly Cystic and partly Solid.

In striking contradistinction to the above case, which is one of general enlargement—a parenchymatous goitre,—is this case of Elizabeth S—, an example of an adenomatous tumour of the thyroid gland two thirds solid and one third cystic. In this patient the symptoms were very marked. There was some difficulty in breathing, but still more in swallowing. When this woman, who is thirty-two years of age, first came under my care there was really little to be seen. The right half of the thyroid gland was, however, enlarged, and just to the right of the middle line there was a hard, firm lump only slightly prominent; when you looked at it you scarcely saw anything, but it

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felt very much harder than the rest of the gland. The patient had some considerable dyspnœa at times, especially when walking upstairs, and when exerting herself to any extent. But her chief difficulty was the great dysphagia from which she suffered, and the fact that food often stuck in her throat. From these symptoms, and feeling the distinct definite hardness of the lump, I had very little hesitation in coming to the conclusion that we had to deal with an adenomatous tumour of the thyroid gland, situated somewhat deeply in the gland, and presenting more on its posterior than on the anterior surface. I therefore operated, and made, as I generally do in goitrous cases, a transverse incision; such an incision has the advantage of being placed low in the neck, and thus the scar is more concealed from view. I divided the deep cervical fascia, which was very strong in this case, and immediately found the explanation of the dyspnœa and dysphagia. The tumour, which had insinuated itself between the trachea and the œsophagus, had pushed the trachea markedly to the left, and the œsophagus somewhat to the right of the midline; the tumour pressed on the posterior yielding non-cartilaginous surface of the trachea, and also on the soft yielding œsophagus. The operation was of a very simple nature, merely the shelling out of the tumour after incising the superjacent thyroid substance. The patient was kept in the hospital only ten days, and was then discharged cured. It is now three weeks since she was operated upon, and her troubles have now completely disappeared. You will see the great point of difference between this case and that of the child. The child had that hard, tense, parenchymatous goitre which gave her pain when handled, but she had practically no pressure symptoms. This patient, with an enlargement nothing like so large as in the former case, had marked pressure symptoms because her tumour was a non-symmetrical one, and had insinuated itself between the trachea and the œsophagus. In this case of adenomatous tumour of the thyroid neither iodine nor thyroid extract was given. It may be imagined that iodine is only of value in cases of parenchymatous goitre, and is of no avail in instances of adenomatous tumours, but this is not always the case. It is not at all uncommon to have cases of adenomatous tumour—I have seen two or three already this year—associated with a good deal of parenchymatous enlargement of the gland. In one case especially there was an adenomatous tumour, about the size of a walnut, in each lobe of the thyroid gland, and also a considerable amount of parenchymatous enlargement. The first time I saw the patient I could not make out the character of the tumour at all, but after the patient had taken iodine for some twelve days the parenchymatous part had shrunk so much that the two tumours, one on either side, stood out prominently. In these cases of adenomata of the thyroid it is advisable to give the patients iodine for a week or ten days, so as to cause the absorption of any parenchymatous enlargement, and thus cause the tumours to stand out much more prominently than they would otherwise do. While on the subject of goitre I may mention that one has always to be on the look-out especially for intra-thoracic goitre. I have only had personal experience of two or three such cases. In one case I certainly came somewhat near losing the patient. A servant girl, sent by Dr. Smith of Chesham, came to the hospital with a good-sized goitre, partly adenomatous and partly parenchymatous, and complained of symptoms of intense dyspnœa. As the adenomata were only small, and the dyspnœa was considerable, I removed the right half of the thyroid gland. The hæmorrhage was considerable, and the patient, who had been throughout the operation very congested, all but collapsed from the hæmorrhage, and had to be transfused with normal saline solution. To my great regret the operation did no good, or at all events but very little benefit resulted.

On returning home the patient had many very violent paroxysms of dyspnœa, in one of which she nearly died. She was therefore sent to me again. Then it occurred to me that, as there was marked dyspnœa, there might be some deep-seated tumour in the neck which could not be felt. Accordingly I began to operate with the object of removing the larger portion of the left lobe of the thyroid, and it was only when I was somewhat near the completion of the procedure that I found a marked intra-thoracic prolongation quite behind the sternum, whence I enucleated a hard goitrous tumour about the size of a turkey's egg. All symptoms of dyspnœa at once disappeared, and the patient made an excellent recovery, except that when last

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I saw her there was some impairment of movement of the left vocal cord. It is, therefore, well to be on the look-out for the possibility of intra-thoracic goitre.

Case 4.—A Case of Lingual Dermoid.

The next case, that of Sarah R—, is not a very rare one, and I show it because in this case a mistaken diagnosis, as not infrequently happens in this condition, was made. The girl is twenty-three years of age, and, as you will see, she has a swelling between the chin and the hyoid bone. When she opens her mouth and raises her tongue you find a tense cystic swelling underneath the tongue in the floor of the mouth. These tumours used to be described in the older books as dermoid cysts in the floor of the mouth, and it used to be a question as to how they originated. We now know that they spring from the thyro-glossal duct. I have not time to enter upon a full description of the thyro-glossal duct. The tongue is developed in two parts, the anterior two thirds of the tongue and the posterior one third being developed separately. These two segments meet, and between them there is the duct called the thyro-glossal duct. The upper end of this duct is what becomes the foramen cæcum, and the blind lower end of the duct forms the isthmus of the thyroid body. The thyro-glossal duct is at first a tube lined with epithelium, which reaches from the foramen cæcum to the uppermost rings of the trachea. When the hyoid bone is developed it divides this duct into two parts: the duct above the hyoid is situated in the tongue, and is called the lingual duct; the portion of the duct below is called the thyroid duct. Thyroid dermoids are developed in the thyroid portion, and lingual dermoids are developed in the lingual segment of the duct. I do not know why it is—I have seen, I think, ten of these cases,—but these dermoid cysts scarcely ever begin to be manifest until after puberty, and when they do begin they often grow with the greatest rapidity. This girl tells us that it is only eight months since she first noticed any lump at all in her mouth, and now she has got a great mass on the floor of the mouth, which is a hindrance to swallowing and even to respiration. She went to another hospital with this cyst bulging between the chin and the hyoid bone—these lingual dermoids always do present both in the submental region and in the floor of the mouth. The tumour was incised, and she was told it was an abscess because some "porridgy" dermoid stuff came out. The skin incision soon healed, and now she has got the lump as big as formerly. To-morrow I intend to remove it, and the operation will be somewhat more difficult than in the generality of these cases, because an incision has been made already, and there has been some leakage probably of dermoid *débris* around the opening in the cyst. A vertical incision should be made from the chin to the hyoid bone, and one must keep in the middle line of the tongue, where there are no vessels separating the genio-hyoglossi muscles, between which the cyst is placed. I should add that there is one thing which it is important to guard against, and that is, do not operate from the mouth. If you enucleate the cyst from the mouth you leave a big cavity, which is bound to be infected from the mouth and is badly placed for drainage.

(*To be concluded.*)

Soluble Salts of Manganese.—Recent experiments have afforded increased evidence that manganese possesses useful qualities in the treatment of debilitating diseases associated with anæmia. It appears, however, that the nature of the combination in which the manganese is employed exercises an important influence upon the therapeutic effects. The preparation known as manganese dioxide, which has been used to some extent in the past, and often in a very impure condition, is quite insoluble in water, and only very slightly soluble in dilute acids. Its administration, therefore, has frequently been unsatisfactory. Some experiments conducted in the Wellcome Chemical Research Laboratories, with a view of preparing a soluble salt of manganese of a stable character, have proved highly satisfactory. As a result of these investigations Messrs. Burroughs Wellcome and Co. have been able to manufacture, and are now issuing, a soluble citrate of manganese. In addition to this preparation they are manufacturing two soluble compounds of manganese and iron. In the treatment of anæmia in which iron alone has failed, these new compounds appear to be admirably adapted to the needs of those practitioners who, instead of passing directly to the use of manganese, prefer to give intermediately a mixture of the two metals.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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ON THE OPERATIVE TREATMENT OF FRACTURES.

Delivered at Guy's Hospital, May, 1903.

By W. ARBUTHNOT LANE, M.S.,

Surgeon to Guy's Hospital and to the Hospital for Sick Children.

GENTLEMEN,—I propose to start by telling you what methods I would advise you to adopt when operating on simple fractures. I will make no apology for entering into the details of the operative procedure in this particular condition, for two reasons. The first is that the bruised and lacerated parts are infiltrated often very extensively with blood, and are in a condition of very considerable inflammation and tension, large bullæ being frequently present. The vitality of the tissues is also enormously depreciated by the presence of the effused blood and by the inflammatory products. Such a state renders any wound made into this damaged part exceedingly liable to infection, and the introduction of any septic organism is very likely to be followed by more or less disastrous results. The second reason for extreme caution is that, in order to leave in the wound such a large foreign body as a screw or thick silver wire, it is necessary that asepsis shall be much more perfect than it is in wounds in which no foreign body is permitted to remain. The absence of sufficient precautions explains the trouble which many surgeons experience in these operations.

We will assume that the fracture has occurred in a subject in whom it is necessary, or who is anxious that no mechanical disability or no pain should follow the injury; also that the fragments of bone cannot be placed or retained in accurate apposition by any process of manipulation, or of extension, or of fixation in splints, casings, etc. We have had enough experience now to know that, providing the fragments have become displaced from one another, whether the fracture be spiral,

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transverse, or oblique in direction, the amount of displacement, overlapping, or alteration of the axes of fragments on one another varies with the hæmorrhage into and swelling of the lacerated soft parts which surround the bone or bones and form ties in its length. We also know that if these soft parts are allowed to remain in their shortened condition for any time the amount of force required to restore them to their original length is necessarily very great, and varies directly with the length of the interval following on the fracture.

As to the time that should elapse between the receipt of the injury and operative interference, I would advise you, in the first instance, to have the part radiographed in at least two planes crossing each other at right angles. You will find that it is generally possible to obtain two photographs at right angles to one another, one showing a minimum of displacement because the rays have passed through the overlapping fragments of bone, and the other the deformity at its worst, the rays having passed at right angles to the plane of displacement. Having done this, proceed to get the skin of the part as clean as possible, and do not confine your attention merely to the neighbourhood of the fracture, but if the injury is one affecting an extremity see that the surface of every portion of it is rendered as clean as it is possible to make it.

In the case of the leg this is particularly necessary owing to the large amount of dried-up epidermis that envelops the heel, sole, and toes. The great thickness of this material which requires to be removed, especially from the feet of the working classes, is sometimes very surprising. After washing the part with soft soap and hot water, apply to the foot a large hot boric acid compress. This can be replaced after a few hours, and the macerated epidermis thoroughly removed. This process may require to be repeated several times.

Then the whole leg should be cleaned with soap, water, and spirit, the skin shaved, and a compress of 1 in 40 carbolic lotion applied for several hours before the operation. When the patient is put on the operating table the compress is removed, the limb being placed upon sterilised sheets or towels, and the skin is again carefully washed with a germicidal solution. This cleaning process having been thoroughly effected, fresh sterile cloths are applied beneath the limb and over the trunk, etc., so as to isolate the limb from the rest of the body. The requisite instruments have been already prepared. They consist of ordinary carpenter's screws in several lengths, and in gauges which by experience have been found most suitable for perforating such a dense structure as the compact tissue of long bones. Roughly speaking, the gauge must be a moderate one. If the calibre is too small there is always a danger of the screw breaking or twisting. At the same time it is inadvisable to use a screw of an unnecessarily great calibre. I would advise you to have a large assortment of screws, as nothing is more annoying than to find you have to use a screw that is too long and projects beyond the bone; while the possibility of the thread not biting the distal compact tissue sufficiently is even more vexing. Therefore take care that your supply of screws is ample.

Have also virgin silver wire in varying thicknesses. The calibre of the wire necessary to stand the torsion or strain exerted upon a junction of the fragments of the femur by the leverage action between fragments, which is very great indeed, must be considerable. On the other hand, that necessary to retain the fragments of the fibula securely in apposition need not be very thick.

I have operated on a good many ununited fractures of the humerus, femur, or tibia which have been previously attempted without success, and I have been amazed at the hopelessly ridiculous character of the loops of silver wire that I have found embedded in the false joint. I would advise you as a general rule to use the thickest wire you can within reason. Again, the wire must be made of perfectly pure silver; otherwise it stands less strain when twisted. The only objection to this is the fact that this wire costs about ten shillings an ounce, and much is liable to be wasted at the operation.

Before using the wire it should be cut up into suitable lengths and straightened. It is then heated to a dull-red heat in a flame and allowed to cool completely before it is placed in the solution with the other instruments. By this means the wire is rendered very much more pliable, and it is possible to apply it with considerable accuracy to the surface of the bone, any slack forming a curve being avoided, and the fragments being retained as accurately, firmly, and immovably as possible on one another. I need hardly impress

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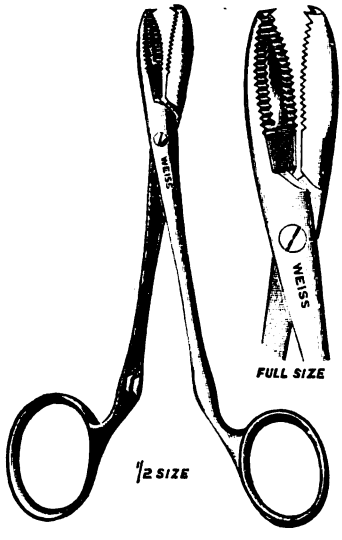
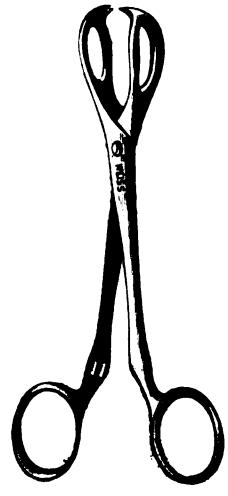
on you the importance of this added pliability, and of the advantages it affords the surgeon. The next instrument of importance is the pressure-forceps, or, as they are commonly called, Spencer Wells's forceps. It is very necessary that these shall exert upon the tissues included in their bite a force sufficient to obliterate the lumen of the vessels contained in them by breaking through the intima. By far the large majority of pressure-forceps are useless for this purpose. They merely control the circulation while they are attached, bleeding continuing on their removal unless a ligature be applied. The disadvantage of the use of ligatures is that the bleeding-point is frequently situated deeply between the bones, and the efficient application of a ligature may necessitate the introduction of the fingers into the wound—a circumstance which must be avoided at all costs if you wish to obtain a successful result. I therefore employ forceps with stout, well-tempered shanks and with accurately fitting blades. To ensure that the ends of the blades shall grip and include the soft parts into which they may be pushed, I have had these pressure-forceps made with three sharp teeth, two being on one blade and including the third on the other when approximated. The presence of these teeth adds greatly to the efficiency of this instrument. Fig. 1 represents such a pair of forceps. The ability of controlling hæmorrhage is an exceedingly important consideration, since the amount of blood that may be lost during such an operation is considerable. This is particularly the case if an interval of some weeks has been allowed to elapse since the receipt of the injury, and in operations for badly united and ununited fractures.

Fig. 1.



When the incision has been made it is absolutely necessary to isolate the skin from possible contact with the wound, or with the instruments used in it. This is best done by attaching the edge of a sterile towel or pad along each margin. By this means the wound is completely isolated, and it cannot be infected from the patient's skin.

Fig. 2.



The pad or towel can be fixed quickly and retained accurately by means of some forceps which I devised for this and other purposes. Fig. 2 represents this instrument. It is possessed of fenestrated blades, which form an olive-shaped head. They are, like those represented in Fig. 1, fitted with teeth which are long and sharp. These hold the margin of the wound and the towel or other sterile cloth, and when held by assistants the edges of the wound are separated to the utmost, and any operative procedures on the structures in it are greatly facilitated. The same forceps are very serviceable when it is necessary to control some bleeding which for some reason or another cannot be stopped with pressure-forceps, the form of its extremity rendering the application of a ligature comparatively easy, it being impossible to include the end of the forceps in the ligature.

Provide a number of strong steel elevators, sequestrum and lion forceps in various sizes, and Peter's bone-forceps. These forceps are very useful

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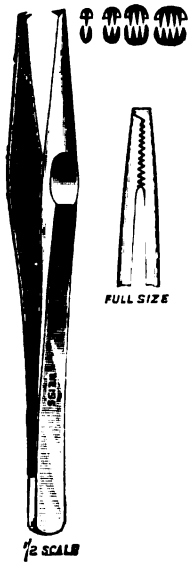
in holding the fragments of a large bone accurately and securely in apposition while they are being perforated by the drill on which the screw or wire is being introduced. To depend on the hand alone is often very exhausting, and any movement, such as changing the hands or the character of the grip, may spoil the results of half an hour's toil. Besides that, the presence of hands whose epidermic covering has been disturbed by pressure near the wound is always a danger, and should be avoided as much as possible. A number of drills of various sorts and sizes, screw-drivers, large, strong, broad-bladed forceps for holding and straining on the wire and twisting the ends into a secure loop, and pliers for cutting the wire, are necessary. The form of the dissecting forceps is very important, since, besides holding skin edges during suturing, they are useful in tearing away lacerated soft parts and in cleaning the broken surfaces of the fragments.

I devised some which I have found most useful. They are made in several sizes, and are represented by Fig. 3. They possess sharp terminal teeth varying in number with the breadth of the blade. These enable one to hold a skin edge or any tissue securely so that it cannot escape from their

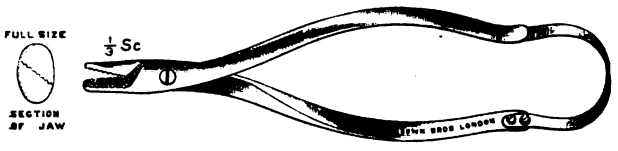
Fig. 3.



grip, as so often happens with the ordinary dissecting forceps. Then the blades are grooved transversely for a length, and are so formed that these surfaces come into accurate apposition when the forceps is closed. This is for the purpose of holding a needle, thread, or horsehair when the wound is being closed.

A good needle-holder must also be provided, since it is important that the needle should not be fingered. There are many varieties, the large majority being provided with a catch. The form I prefer is shown in Fig. 4. The peculiarities it

Fig. 4.



presents are that it has a spring which keeps the blades apart sufficiently to allow of the needle being grasped easily and quickly and held securely.

The plane of approximation of the blades is neither vertical nor transverse, but intermediate between these. The advantage of this obliquity is that a curved needle can be held and inserted into the distal margin of the incision without rotating the hand excessively, as must be done when the grasp is horizontal. The objection to this needle-holder is that it requires a little practice in its use, since most beginners cannot do two things at the same time. They can hold the needle securely till they commence to insert it into the flap, when the grasp of the hand relaxes involuntarily. When this initial difficulty has been overcome its use is exceedingly easy and satisfactory. If the needle is a large one it is grasped high up between the jaws, while one of small calibre is held near their extremity.

Have plenty of fishing-gut and horsehair soaking in a lotion of 1 in 20 carbolic, and a reel of No. 1 silk should be boiled with the instruments on the chance of its being required to ligature some vessel that cannot be dealt with otherwise.

As to swabs and dressings, I prefer them sterilised and dry. As, however, you may be unable to obtain such that you can rely on absolutely, you can make use of pads that have been boiled in water and squeezed as dry as possible in a sterilised towel. You can also, if you please, employ as a dressing cyanide gauze which has been sterilised in carbolic lotion, and then free from the acid by soaking in boiling water if you cannot obtain dry sterile pads.

No germicidal solution should be introduced

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into the wound, since it only serves to irritate the tissues and depress their vitality, and render infection of the wound more possible.

Now proceeding to the operation itself, the first thing to consider is the locality of the incision or incisions.

Two factors must govern your choice of incision, one being the possibility of avoiding damage to important structures, and the other the ability of making a very long incision, as upon the length of the incision depends the facility and success with which the operation can be performed, and the consequent freedom from risk of infection.

The secret of success in these operations is to have a wound of sufficient extent to allow you to expose the overriding ends of the fragments as completely as possible, so that you get at them readily with your instruments. I do not think I can do better than describe in sequence the details of the operative procedures necessary to re-establish continuity in spiral fractures of the femur, and tibia, and fibula, and in Pott's fracture. After doing this I can consider more briefly the treat-

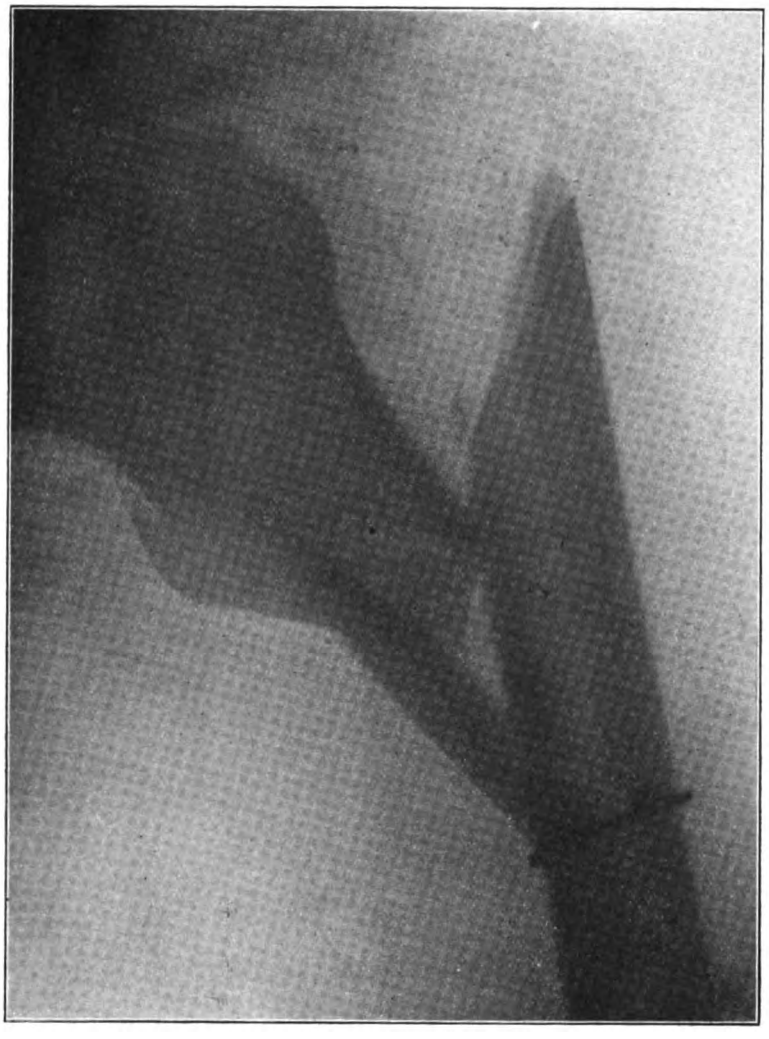

FIG. 5 shows the relative displacement in a transverse plane of the fragments of the femur in a case of spiral fracture. Their position differs somewhat from that which existed originally, since the lower end of the upper fragment is retained in contact with the lower by an encircling loop of wire. (For the loan of this and the succeeding block we are indebted to the Editor of the 'Practitioner.')

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ment of fractures of the upper extremity. In the first instance—namely, fracture of the femur—if backwards, and outwards upon the upper, the upper passing downwards, forwards, and inwards.

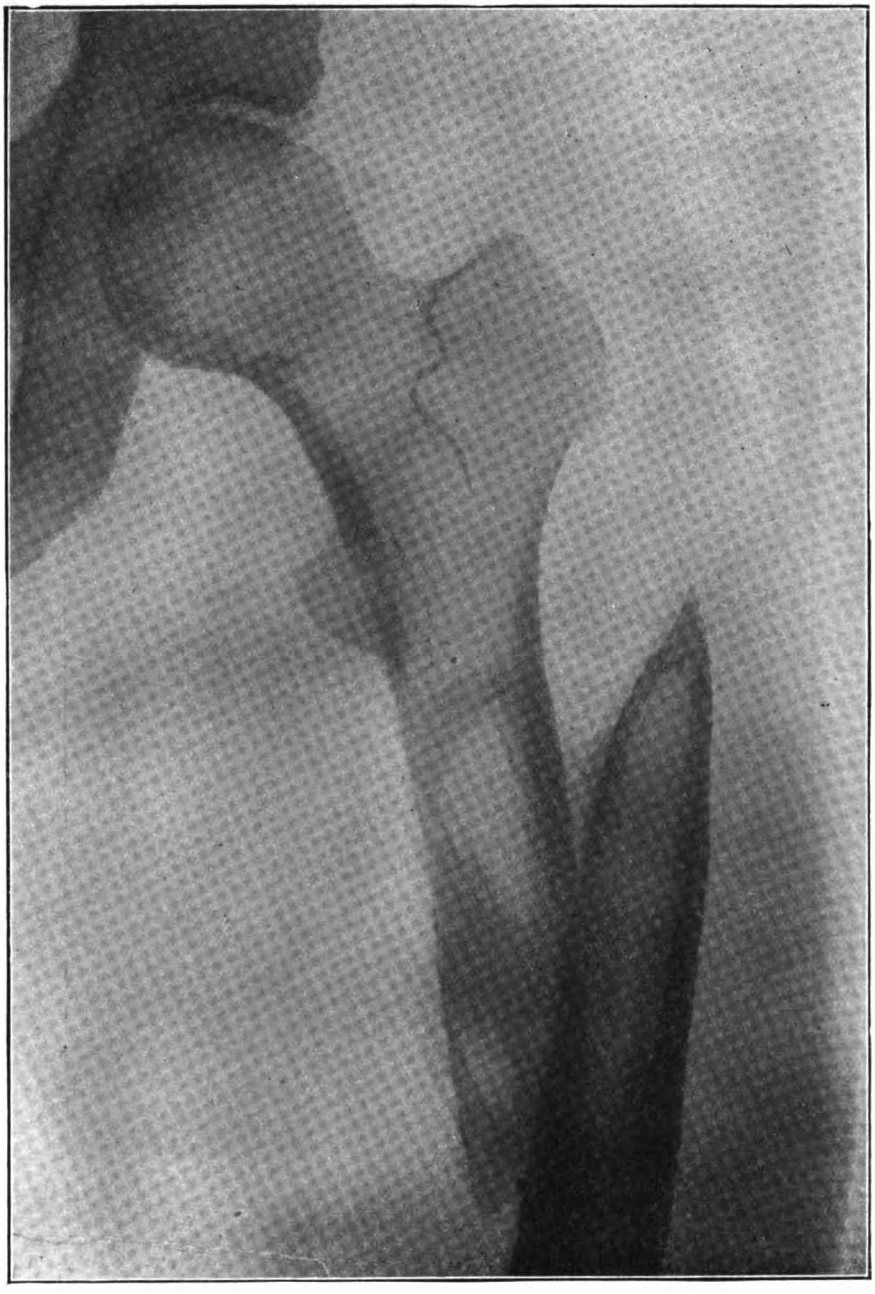

FIG. 6 shows the relative position of the fragments of the femur in a case of spiral fracture which had been treated by splints. The upper extremity of the lower fragment has lost through absorption a little of its original length. As in Fig. 5, the lower fragment lies outside the upper.

the patient is an adult you know that the lower fragment is almost certainly displaced upwards, In the case of a child, where the fracture may result from rotation of the thigh inwards, the displacement

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of the fragments is occasionally reversed, as can be seen in the radiographs shown, which represent the condition before and after operation. In it the lower fragment has passed upwards, backwards, and inwards, and the upper downwards, forwards, and outwards.

The mode of displacement makes no difference whatever in the incision.

The patient's trunk is turned a little towards the opposite side, and is supported in that position by sandbags in order that the outer aspect of the damaged thigh is most readily exposed.

If the fracture be in the position indicated in Figs. 5 and 6, an incision is made along the whole length of the thigh externally in a line corresponding to the interval between the extensor and flexor muscles. These muscles are divided or separated along the external intermuscular septum till the fragments of the femur have been freely exposed. Their ends are then freed from the surrounding soft parts till a sufficient length of each can be manipulated. The fractured surfaces are carefully examined, and anything that can get between them forced aside and retained there.

While an assistant exerts traction on the leg the fragments are grasped in the blades of powerful forceps, and as the lower fragment is brought downwards upon the upper you endeavour by means of a broad, strong steel elevator to manipulate the moving fragments forcibly approximated by the grasping blades of the lion or other forceps, so as to bring the fractured surfaces into accurate apposition. It is simply a question of time, perseverance, and careful and dexterous manipulation, both on your part and on that of your assistants. Having obtained accurate apposition, apply a pair of Peter's forceps in such a position as will interfere as little as possible with the drilling of the holes and the passage of the wire or screw.

You must decide as to what particular medium you will adopt. If the fracture is a long spiral one you had better use a screw perforating the centre of the junction. Remember in drilling the hole for the screw that the lumen of that portion of it that is occupied by the barrel must be greater than that which is filled by the thread. If this precaution is not observed you are very liable to fissure and break up the proximal fragment. Make a hole above and below, and pass through each a loop of very strong virgin silver wire. Having rendered them as tense as possible, twist them up securely, making sure that you have not strained the wire excessively and broken it. Having done this, and having stopped all bleeding-points, bring the flaps of skin together temporarily by means of reverses of fishing-gut in such a way as to enable you to retain its edges accurately together with continuous sutures of horsehair. This apposition of the skin edges should be absolutely perfect, and is by no means the least important portion of the operation. When it has been effected the fishing-gut sutures are removed and large sterile pads are applied.

The most satisfactory splint is that called a double Thomas's hip splint, since it takes all strain off the junction, and it enables the nurse to move the patient about freely, and to attend to comfort and cleanliness without risk to the fracture.

The dressing should not be touched for two or three weeks, when the horsehair sutures can be removed.

After six weeks have elapsed the splint can be removed and the patient permitted to move the leg freely in bed. As soon as its strength is regained he can get up and use the limb. There need not be the slightest anxiety on the part of the surgeon as to the stiffness or rigidity of joints which accompanies the very imperfect junction obtained by the use of splints alone.

(*To be continued.*)

ACCORDING to Dr. W. Coleman, urotropin is a product resulting from the action of formaldehyde on ammonia. Toxic symptoms are not infrequently noted under a daily dosage of fifteen grains, and individuals differ greatly in their susceptibility to it. According to Keyes, this susceptibility varies in the same individual at different times. Urotropin is quickly absorbed and eliminated, the rapidity of its complete elimination depending upon the dosage. Apparently urotropin is not ordinarily decomposed in the blood. Formaldehyde cannot be demonstrated in the urine of persons taking urotropin, yet clinical experience shows that it possesses a distinctly antiseptic action on the urine. One observer has maintained that acid urines apparently produce a partial decomposition of the urine. Whatever may be the action of urotropin, it is the most powerful urinary antiseptic in our possession.—*Medical Record*, June 13th.

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LECTURES ON SOME AFFECTIONS OF THE HIP.

Lecture III.

ON CONGENITAL DISLOCATION OF THE HIP.

By A. H. TUBBY, M.S., F.R.C.S.,

Surgeon to the Westminster Hospital; Senior Surgeon, Evelina Hospital for Sick Children, and Consulting Surgeon to the Hip Hospital, Sevenoaks.

Gentlemen,—To-day I propose to continue the subject of the affections of the hip-joint, and to deal more particularly with congenital dislocations.

It would be well, however, to define the term congenital dislocation, for it is not every hip which is found displaced immediately after delivery which can be described as congenitally dislocated. Some displacements are undoubtedly due to trauma during parturition, and arise from violence in delivery in breech presentations. That such accidents may occur is shown by experiments of Melicher at Vienna upon women who had died undelivered. In six trials he found that with the fingers or hook it was possible to produce a dislocation, and in two instances in which the displacement was unilateral, the dislocation was in that hip to which greater traction had been applied. Reduction in these cases was effected with the greatest difficulty. Cases such as these are acquired and not congenital dislocations, because on examination it can be shown that the joint is perfectly developed and violent displacement has occurred.

In comparison with other joint affections congenital displacements are not very common; and to those engaged in ordinary surgical practice they may appear rare, because they drift into the hands of specialists, who see the majority of them. The affection is seen seven times as frequently in females as in males. The reason for this is not known, but it seems to descend in the female line. The affection is both unilateral and bilateral, and the proportion of cases so affected is about equal.

Congenital displacement of the head of the femur is an affection which dates from intra-uterine life. It may be observed immediately after birth, and is one in which no signs of traumatism are necessarily present. In fact, the joint may be said to be in a state of unstable equilibrium due to the presence of a misshaped or partially deficient acetabulum, and the movements of parturition, especially in the cases of breech presentation, are sufficient to shift the head from the position in which it should normally lie. At birth this displacement, which may be only a partial one, is transformed into a complete one.

It is well known that the deformity is hereditary. There is one case recorded of a man who had two daughters, the younger of whom had a unilateral displacement and the elder one a double displacement. Another case was that of a father whose left hip was displaced, and he had a daughter whose left hip also was affected. A third case was a mother, well formed, who had ten children, amongst whom were two daughters afflicted, one with double and the other with single luxation. The granddaughter had nine children, and one of these had a unilateral dislocation. It is true that these examples are rare, and must be sought for, but they suffice to establish the hereditary factor.

In regard to the pathological anatomy, it is well to describe in simple terms the method of development of the hip-joint. The earliest condition of the pelvis and femur is that of a more or less vertical plate of mesoblastic tissue representing the side of the pelvis, and lying in contact with it at one of its extremities is a rod of mesoblastic tissue which ultimately becomes the femur. Gradually, from the upper end of this rod, which has now become cartilaginous, the head of the femur emerges and assumes its shape and takes its concrete form, but it still lies against the flat surface of the pelvis. At the spot where the head is in contact with the pelvic cartilage the normal acetabulum will form, and in the following way:—The pelvis thickens at that place, and the acetabulum grows up around the head of the femur so as to embrace it for two thirds of its circumference. It should be remembered that the pelvic cartilage consists of three distinct portions, which meet and unite at the bottom of the acetabulum.

Congenital dislocation is associated always with changes in the acetabulum, but not, in the developmental state, with alterations in the head of the femur. The latter, so far as our observations go, is normal at birth.

The alterations or deficiencies of the acetabulum are as follows:—In some cases it is partially wanting, and in such there is found in the place of the coty-

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loid cavity a circular surface more or less flat, about half an inch in diameter, and covered with thick fibrous tissue. At the posterior border of the surface there is sometimes found a small crest which corresponds to the posterior superior border of a normal acetabulum, but this is a rare condition. More often there is found during the course of an operation, where the normal acetabulum should be, a convex mass of bone. I have found such in a case. The clinical importance of recognising this latter-named condition is twofold. The position of the artificial acetabulum is clearly indicated, and there is probably sufficient thickness of bone to form a cup for the head of the femur.

In contrast with the conditions which we have just mentioned are those instances in which the cotyloid cavity is present, but defective. Sometimes it is found nearly, but not quite of normal development, and not of circular shape. The most frequent form it assumes is that of an isosceles triangle with its base upwards and forwards, and there is no doubt that this appearance arises from suppression of the ischial portion of the Y-shaped cartilage. In some cases the cotyloid cavity is of normal circular shape, but deficient in depth and deficient in the height of its borders.

We may sum up the abnormalities of the acetabulum as follows:—In depth it is frequently shallow, and scarcely large enough to admit the tip of the index finger; in shape it is somewhat circular, but more often triangular. Sometimes it is entirely absent, and sometimes its place is occupied by a convex mass of bone. Frequently the postero-superior border is deficient.

The subsequent changes in the part which take place are entirely due to adaptation. At birth the head of the femur is, as we have before stated, generally normal in shape; but probably from the time of birth, and certainly from the moment when the child begins to walk, it undergoes the following changes:—The head loses its circular outline and becomes flattened on one aspect. Thus, if the dislocation be dorsal, the anterior aspect is flattened; and if the dislocation be anterior the reverse occurs. It also becomes oval in shape from above downwards, and sometimes irregular. In adult life it is frequently mushroom-like, and in old age it may disappear entirely. Such an instance was found in a woman æt. 65. Occasionally the growth of the head is retarded in such a way that one belonging to a child of ten years will be as large only as that of a normal five-year-old child, and as age progresses the disparity becomes more and more marked. In other cases the neck becomes shortened and altered in direction. As age advances it becomes less obtuse, and finally horizontal. Further, it is atrophied, and appears to be merely an isthmus connecting the head with the great trochanter.

An important change is that the neck undergoes version in such a way as to become convex anteriorly or posteriorly. Carefully considering these changes in the upper extremity of the femur, it is quite evident that treatment to be effective must be begun before the child has walked; and the later the case is left, the less is the prospect of a satisfactory result, on account of the changes in the bony parts. Even when it is possible to replace the head of the femur, the version in the neck causes the foot to turn either outwards or much inwards; but doubtless this can be overcome by an osteotomy of the femur.

As to the ligaments, the round ligament has been found thick and solid, or elongated and attenuated, or absent in about 83 per cent. of the cases. The capsular ligament is in most cases elongated; in some it has undergone remarkable thickening, and perhaps it has served to partially support the weight of the body. In other cases it is dilated, and there is a fold of it between the bones. Sometimes it has a curious hour-glass shape, and is obliterated in the middle.

The changes in the muscles may be briefly summed up as follows:—Those which pass from the pelvis to the great trochanter—that is, the abductors and external rotators—are lengthened, whilst the adductors are shortened. The ilio-psoas undergoes a change in direction: instead of lying over the head and neck of the femur it comes to pass more and more internally, and it is situated over the anterior border of the cotyloid cavity; and finally, as the head becomes more and more displaced, it passes over the posterior border. This new position may account for the curious hour-glass shape of the capsular ligament, which is a very definite obstacle to reduction.

Direction of the Displacement.—In the majority of cases it is superior and posterior, but as the subject is more closely studied the number of anterior

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displacements is found to be considerable. I have observed during the past eight years no less than seventeen.

In the case of dorsal dislocations, there may be found hollowed-out spaces where successive and partial lodgments of the head have taken place. And there was the classical case of Paletta, in which there were three distinct smooth surfaces, indicating the three positions occupied by the head of the femur at different times during life.

Is the spontaneous formation of a new joint possible—if it be a true joint? No! If it be an osteo-fibrous joint? Yes! But the chief obstacles to the formation of a nearthrosis capable of sustaining the body-weight are as follows:—First of all the downward thrust, tending to push the head of the femur further out of place; secondly, the interposition, between the periosteum of the ilium and the head of the femur, of the thickened capsule and of the elongated round ligament; and, thirdly, the constant adduction of the femur in such a way that its head loses its full contact with the iliac bone. In all cases of congenital dislocation there is probably a new cavity sufficiently large to receive the head of the femur, but unfortunately this is not osseous, and the bone is particularly wanting at the upper and posterior part, so that the head continues to slip upwards.

Symptoms.—The mode of walking is quite definite. In a unilateral case the patient raises himself somewhat on his toes, and he inclines the upper part of the body towards that foot which is chiefly supporting the weight. Then the other foot is carried forward. At that moment the trochanter of the limb, on which the weight of the body falls, can be felt to telescope upwards towards the crest of the ilium, and then when the weight is taken off that side it resumes nearly its former position. So that the gait is waddling; but when the patient is running this is not so marked. Much lordosis is present on account of the disturbance of the body equilibrium. As the patient stands it may be noticed that the hands reach much lower down on the thighs than in a well-proportioned individual, the muscles of the lower extremity are small and ill-developed, and the appearance is exaggerated by the want of proportion between the size of the body and lower limbs. The pelvis itself appears broader than is usual. When the child stands, the great trochanters are angular and unduly prominent, and they are displaced upwards. The gluteal muscles form a cone with the apex at the great trochanter, and the heels are rotated inwards and the toes turned outwards.

On placing the patient on his back the lordosis disappears, and the distance between the tops of the trochanters and the crests of the ilia becomes less. In all cases, however, the trochanter is above Nelaton's line, and there is shortening of the limb, much of which may be made to disappear by steady traction. When the traction is lessened the head of the femur goes up again, and this movement is known as the "telescope" movement. The negative signs are as follows:—Total absence of pain; no limitation of movement in young children; and little limitation in adolescence.

Diagnosis.—It is essential to be quite certain that the displacement of the head of the femur is really congenital and not due to severe traumatism at birth. One must also be careful not to accept too readily the assertion of the parent that the child has had a fall, for which the nurse is often blamed. It is difficult to imagine that a mere fall will cause a dislocation of a hip without signs of bruising and extravasation beneath the skin. If the affection is bilateral it is easier to form an opinion than when it is unilateral. Confusion has arisen between congenital dislocation and coxa vara, but in the latter affection the great trochanter cannot be drawn down by traction of the limb, and the head of the bone is in its normal situation. The waddling gait and the prominence of the trochanter in coxa vara, together with the difficulty in abducting the limb, have caused the two affections to be confounded; but the best test of all is the use of the X-rays in a doubtful case, and those which have been frequently called coxa vara have been shown by these means to be congenital dislocations.

On account of the waddling gait in bilateral cases of congenital dislocation and in pseudo-hypertrophic paralysis, difficulties in diagnosis have arisen. The point is to find out the position of the head of the femur. And in pseudo-hypertrophic paralysis there are hypertrophy of the gastrocnemius and the extensors of the thigh, and atrophy of the lower part of the pectoralis major, the latissimus dorsi, and the teres major; while the peculiar movements of the child when raising him-

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self from the horizontal to the vertical position in this affection are quite characteristic.

A diagnosis has frequently to be made from coxitis, especially when the head of the femur has undergone pathological dislocation; but a careful inquiry into the history of the affection—as to its onset, its progress, the presence or absence of sinuses, the painful limitation of the movement of the joint, the existence of adduction and flexion—will serve as means of distinction, while the movements in congenital dislocation are quite free and painless.

Occasionally paralytic dislocation is met with, and if the child has not walked the difficulties are great, and there can be no doubt that many so-called congenital displacements are really paralytic. Confusion is the more likely to arise because the caput femoris is in both cases very mobile, and moderate traction brings it back into position; but the muscles should be very carefully tested for signs of degeneration, and by careful and skilful movements the head of the femur can be completely replaced in the acetabulum in a paralytic case, but not often in a congenital one.

Treatment.—The object of all treatment should be to completely remove the deformity with the substitution of a sound joint for the loose and telescopic one, and the removal of the waddling gait. The difficulties in the way of perfect treatment we have already enumerated, and we may briefly repeat them. These are—the ill-developed or shallow acetabulum; its irregular shape and diminutive size; the misshaped head of the femur; the version of the neck; the condition of the capsular and other ligaments; and the contraction of muscles.

Many methods have been employed, but three at least deserve serious notice. These are—(1) prolonged recumbency with extension; (2) reduction by the open method, as practised by Hoffa and Lorenz; (3) forcible extension and reduction by manipulation according to the method originally advocated by Paci, and recently modified by Hoffa and by Lorenz. But it must be at once confessed that no method gives perfect results.

1. The oldest method is that of complete extension in the recumbent position, but in none of the cases was a complete cure effected; and, indeed, the classical case of Buckminster Brown, which was averred to have been cured, had nine years afterwards suffered a relapse. The method has many disadvantages, such as the long duration of recumbency and of the after-treatment, and interference with the general health. At least four years' constant supervision is involved, with much expense, and not infrequently disappointment follows.

Schede has for some years past treated cases under two years of age, in whom no secondary changes have been caused by walking, by simple traction, slight abduction, and moderate lateral pressure on the trochanter by which means the head of the bone is retained in or on the acetabulum. After the child has commenced to walk the prognosis is less favourable. By the end of the second year, and certainly the third, the changes in the joint become so marked that extension alone no longer suffices to reduce the deformity. In such cases Schede employs continuous extension by means of weights for a few weeks or months, and so restores the position of the head of the femur that, with slight abduction and pressure over the great trochanter, the head of the bone may be made to go back into its place, and remain so firmly fixed that pressure upon the sole of the foot no longer causes the head to slip out. With the idea of exciting a firm fibrous or bony growth at the spot where the head of the femur rests, Lannelongue has injected a few minims of a 10 per cent. solution of chloride of zinc from time to time. The practical conclusion to be deduced from this proceeding is that, in order to obtain a cure, some inflammation must be excited; and this is just what the treatment by rest and extension in the recumbent position fails to effect, and hence it cannot hold out hope of a permanently satisfactory result. Indeed, it seems that in order to obtain a cure we must just produce that happy degree of mild inflammation resulting in some fibrous bond between the bones, and not that severe degree of inflammation sufficient to produce absolute ankylosis. And if we could obtain the same result as that which is seen after some cases of arthrodesis of the ankle, in which there is fibrous ankylosis sufficient to allow movement, but still maintaining the joint in a stable position, the ideal result in congenital dislocation of the hip would be obtained.

2. *Treatment by the Method of Open Operation.*—This was first advocated by Hoffa, but his method has now been entirely discarded for several reasons, not the least important of which

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is because of its extreme severity and danger. Lorenz, in place of cutting so widely as Hoffa, contented himself with an anterior incision between the sartorius and the tensor vaginæ femoris. In a work on the subject published in 1897 Lorenz gives details of two hundred cases, but few of these appear to me upon close examination to be unqualified successes. In the earlier operations he met with many difficulties and some disasters. Thus two patients died of sepsis, one bilateral case was followed by ankylosis of both sides, one operation was left unfinished because of the difficulties attending it, in two the deformity recurred, in one the reduction was difficult and prolonged. Three were complete failures because the after-treatment could not be carried out. The remainder of the one hundred cases are claimed as unqualified successes, with four exceptions, in which limited movement and partial ankylosis followed; but it is to be noticed that, however perfect the anatomical results as to the reposition of the head may have been, the functional result was far from being so. It is also remarkable that when surgeons of great operative skill and experience endeavoured to follow in Lorenz's footsteps they met with nothing like the same measure of success. A noteworthy point is this—that Lorenz has abandoned this method and now resorts to a forcible reduction whenever possible; and the ready abandonment of the open method by its great advocate does not speak well for it. I have had some personal knowledge of the open operation, and it is one of considerable difficulty and severity. In one case upon which I operated I found the head of the femur misshapen and retroverted, the acetabulum absent, and in its place a bony crest; the capsule was also large, loose, and lax. It was not possible to deepen the os innominatum sufficiently to form a new acetabulum, but a considerable depression was made by gouging. Within five days of the operation the child developed scarlet fever, contracting it from a patient in the next bed. The whole wound broke down and finally healed by granulation. A complete cure followed, which was not directly due to the mode of operation, but rather to a sufficient degree of inflammation excited by the attack of scarlet fever, with subsequent formation of fibrous tissue. It seems, therefore, that what we should aim at is fibrous ankylosis, and not a new joint.

3. *Forcible Reduction by Manipulation.*—The manœuvres originally practised by Paci, and strongly advocated and successfully practised by Mr. H. A. Reeves, are as follows:—The patient is placed recumbent and the surgeon flexes the leg fully on the thigh and the thigh on the pelvis, thus carrying the head of the femur downwards. The thigh is now abducted fully, and the limb is strongly rotated outwards and then extended fully. If the displacement be anterior, the limb should be abducted and rotated inwards. Paci claims that the head is brought as near to the acetabulum as possible, but it is well to note his *bonâ fides* in the statement that he does not say it is always put into the acetabulum. Afterwards the limb is put up in silica or plaster-of-Paris bandages for a month, followed by continuous extension for three months. Then walking may be commenced on crutches.

Acting on the hints conveyed by Paci's procedure, Lorenz, after much thought and careful observation of the cases operated upon, adopted Paci's method with some modifications. The details of Lorenz's latest proceedings are as follows:—Believing that the resistance of the adductor muscles of the thigh is one of the chief obstacles to drawing the head of the femur down, Lorenz first places them on the stretch by abducting the limb, and then ruptures them by frequent blows of the ulnar border of his outstretched hand. If the operation is called bloodless, it is only so because there is no blood externally. There is doubtless plenty of it effused beneath the skin. The hip is then forcibly flexed, with the knee fully extended so as to stretch the extensor muscles, and a similar process is carried on with the patient lying on his face so as to stretch the flexors. When the movements have been carried out sufficiently vigorously, and possibly violently, the muscles are entirely relaxed, and the outstretched limb can be made to describe nearly three parts of a circle in the antero-posterior plane of the body. As the result of relaxation of the muscles, the top of the trochanter can be drawn well down when vertical traction is made on the limb. An attempt is now made to place the head of the femur in the acetabulum. The operator stands well over his patient; he flexes the hip and bends the knee to a right angle. The thigh is then brought into full abduction, and if this be forcible and complete enough, reduction is said to occur, accompanied by a dis-

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tinct sound. So far it is surmised that the resistance of the anterior part of the capsular ligament may not have been fully overcome. The abducted limb is therefore hyperextended, and plaster of Paris is applied with the thigh abducted to an angle of nearly 90 degrees. The plaster embraces the pelvis, the affected hip, and the thigh, down to the knee. The plaster is kept on six months and then removed, and the hip is gently exercised in the directions of adduction and abduction. Flexion and extension are movements to be practised later with care. The patient may be allowed to walk about if the case be a unilateral one, with the boot on the sound side.

Lorenz's modification of Paci's operation is not above criticism. Very disastrous results have occurred owing to the violence employed. Gangrene, fractures, and persistent paralysis of the sciatic nerve are all on record. It is also difficult to understand how a misshapen and distorted head of the femur can be placed into a small, ill-developed, and partially filled up acetabulum; and even if it does get there, how is it to be retained there when the patient walks? What we require in this procedure is a certain number of cases, to be selected by a committee of experts, carefully examined, measured, and photographed, and X-ray pictures taken in every case before the operation and immediately after the operation, and six, twelve, and twenty-four months subsequently. At the expiration of two years from the time of operation, and twelve or eighteen months after the patient has been allowed to walk about, these cases should be re-examined and reported upon. Cases of no particular age should be taken, but of all ages.

Up to the age of four years I have met with striking success, so far as the immediate results are concerned, by dividing either subcutaneously or by the open method the contracted adductors, sartorius and tensor vaginæ femoris; then, exercising traction on the limb and standing well over the patient, have fully flexed the thigh, vigorously abducted it, and felt it to go in with a click. Afterwards I have kept my patients in plaster for six months to a year, and then fitted an apparatus which allows them to go about with the limb abducted at an angle of forty-five to sixty degrees. But I am not yet convinced that it is safe with these patients, even three or four years afterwards, to go about without some form of retentive appliance. In double congenital dislocation it is advisable to operate on one leg at a time, and when the joint has been rendered stable and firm the second limb is treated. Lorenz states that the operation is most successful in children under four, and such has always been my experience.

As to the merits of the methods, it seems that on the whole Paci's method, as modified by Lorenz, promises the best results. But it is quite evident that these are not as good as they ought to be, and further earnest work is greatly needed on this subject.

Primary Abdominal Pregnancy (Omental).—Dr. K. Witthauer ('Centbl. f. Gyn.,' January 31st, 1903) records the first case in which the embedding and development of an impregnated ovum on a projection of the omentum has ever been described in the human being. The patient was æt. 23 years. She had had two premature deliveries at eight months. After skipping two menstrual periods she began to bleed irregularly, and after another month she came under observation. After careful examination the diagnosis of tubal abortion was made. Under sudden collapse symptoms a laparotomy was done. The peritoneal cavity contained free blood (1½ litre). On the right side an ovarian cyst the size of a hen's egg was removed with the healthy tube. The left adnexa were found healthy and the uterus not enlarged. The rest of the pelvis showed no cause for the bleeding. In the right pelvis an omental projection was the seat of a hæmatoma of the thickness of a finger. This was removed after ligature of its base. Recovery. The removed tube showed no pathological changes, and the ovary was the seat of an ordinary cyst. The careful microscopic study of the omental projection which was amputated showed that the omentum had enveloped the ovum. Blood was found between both. It was not possible to discover villi in direct contact with the omentum, but this was explained by the supposition that the bleeding separated the ovum from its surroundings. —*The Post Graduate*, June, 1903.

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WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

General Pathology of Fever.—I.

Fever is a process characterised by increase of body temperature and disorder of nutrition, and is due to the entrance into the circulating fluids of certain morbid products which are usually of bacterial origin.

Since the introduction of the clinical thermometer into medical practice—a comparatively recent event—the term fever has come to be often used as though it were synonymous with one of its most prominent and constant symptoms, namely, pyrexia.

Such a use is both inaccurate and misleading; for many fevers are or may be apyrexial during some portion or at certain periods of their course. And, on the other hand, pyrexia may be present without fever, as in the case of the so-called cerebral puncture, in various head injuries, in insolation, and after the administration of certain drugs. The presence of pyrexia, therefore, is not in itself sufficient for the diagnosis of fever, nor does its absence at the time of observation exclude that condition. It is an easily recognised phenomenon, and hence convenient as a clinical guide; but its importance is in general very much less than that of the other event which I have mentioned, namely, the disorder of nutrition which invariably occurs in fever.

The older views of fever regarded it as a state of over-action. That theory is interesting only from the historical standpoint. It was supported by no evidence except the observed pyrexia which was attributed to an over-production of heat, the observed increase of the heart's action, and a supposed increase of tension in the whole vascular system. It was, moreover, fundamentally at fault in looking upon fever as a *state*, instead of as a *process* marked by a definite succession of stages.

As Sir John Burdon Sanderson points out, Johannes Müller was the first to recognise it as a reactive process. He compared it to a reflex action, and supposed it to be initiated through a stimulation of the spinal cord by afferent "organic nerves."

Vaso-constrictor nerves had already been discovered by Claude Bernard, and about this time Weber discovered the inhibitory action of the vagus on the heart. Then followed the discovery of Schiff of the existence of vaso-dilator mechanisms. And thus arose the conception of the vascular system as controlled by nervous centres by means of two antagonistic sets of efferent nerve-fibres.

From the idea of the controlling of organic processes by a double innervation from the central nervous system, the one stimulating in its action, the other quelling, thus introduced in physiology, Virchow developed his celebrated view of fever which regarded it as a neurosis—the outcome of a failure of the control normally exercised by the central nervous system not only on the vascular system, but, as he suggested, on all the organic processes of the body. This theory of a loss of control over the circulation and metabolism by the so-called "moderating centres" absolutely reversed the teaching previously accepted of an over-activity, and exhibited the events of fever as the outcome of a paralysis of all the controlling centres. It appeared to receive support from the observation of Traube (1855) two years later that the excretion of nitrogen increases during fever. This could be attributed to the paralysis of the trophic nerves normally controlling katabolic action.

Traube himself, however, regarded the increased breaking down of proteids as being secondary to the circulatory disturbance. He held that the fever-producing agent acted on the vaso-constrictor nerves, causing contraction of the peripheral arterioles, and hence diminished flow of blood to the surface, diminished temperature of the skin, and diminished loss of heat, with the result that the internal temperature rose rapidly. To the increasing difference between the internal and the surface temperature he attributed the rigor of accession. He taught that normally the *discharge* of heat at the surface was *regulated in accordance with* the heat production of the body. The principal event in fever was a disordering of the regulative heat-discharge in the direction of diminished loss, so that an undue amount of heat was retained in the body and the temperature became increased. All the other events were secondary to this.

In opposition to Traube it was shown by Liebermeister that even if the temperature of the surroundings be raised to such a height that no

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heat can leave the body, and surface loss becomes impossible, yet the temperature of a healthy individual does not rise nearly so quickly as in the initial stage of many fevers. The theory of Traube therefore could not be maintained, and Liebermeister came to the conclusion that in fever as in health the temperature is regulated by the *adaptation of production to discharge*, but that in fever the normal or standard to which it is regulated is several degrees higher than in health. By this he meant that the temperature instead of tending to a normal of say 37·5° C. (98·5° F.), as in the condition of health, tended towards a mean of 40° C. (104° F.), or whatever it might be in different cases. And certain evidence seemed to support this view, which was accepted by the late Dr. Hilton Fagge. Thus the temperature of a healthy individual can be increased or depressed by placing him in a bath, and gradually warming or cooling the water.

If the body temperature be by this means increased as much as one fifth of a degree C. (about one third of a degree F.) above the normal the subject will perspire; if it be lowered below the normal by the same amount he shivers. The same observations are found to hold for a fever patient whose body temperature is, say, 40° C. (104° F.). If his temperature be lowered by cooling him in a bath to 39·8° C. he shivers, while if it be raised to 40·2° C. he will perspire. The explanation of this is that it is the change of temperature and not the temperature itself which causes the reactive shivering or sweating. If the alteration be produced extremely gradually, as in the method of tepid sponging in enteric fever, the rectal temperature can be reduced by a whole degree or more of Centigrade (2° F.) without resultant shivering taking place.

Liebermeister, however, went further than was warranted by his experiments when he concluded that, because arrest of surface loss of heat in a healthy individual does not cause such a rapid rise of temperature as occurs at the onset of many fevers, an arrest of surface loss is inadequate to produce pyrexia, and that therefore the nervous control which suffers diminution in fever is one normally exercised by means of regulated heat-production.

Traube's theory, on the other hand, attributing everything to vaso-motor action, regarded the control as one consisting in a nervous regulation of the surface loss of heat.

The view of Liebermeister found support in the experiments of Finkler, carried out in the laboratory of the physiologist Pflüger, on the respiratory exchange of fevered animals, and was endorsed by Cohnheim in his well-known lectures. That of Traube gained adherence from the results obtained by Senator and Leyden, both on animals and in the course of clinical investigation also.

The phenomena of fever, which we must presently consider, may be conveniently grouped under four headings, viz. the disturbance of temperature, alterations in the pulse and circulation, nervous phenomena, and lastly, the disorder of metabolism including the respiratory exchange.

The process of fever itself is divisible into three stages or periods, namely, the period of onset or the *accession*, the *fastigium* or period of continuance, and the stage of *defervescence*. The accession may be abrupt or gradual. When it is gradual the temperature curve usually exhibits a daily remission corresponding to the normal morning fall of temperature in health. The fastigium may be long, or of such short duration as to be almost imperceptible; and the defervescence may occur rapidly, the temperature falling by crisis, or may be more prolonged and show a gradual fall of temperature by lysis.

As regards temperature the fevers are divided into various classes according as the pyrexia is *continuous; remittent*—that is varying more than about 1° C. (2° F.) in the twenty-four hours; *intermittent*, that is, falling to or below the line of normal temperature in the daily remission; or *irregular*, a term which sufficiently explains itself.

The continued fevers have all the stages long. There is a gradual onset, as, for example, in typhoid fever, where in a typical accession the temperature rises about a degree of Centigrade (2° F.) each evening, falling about one third of a degree Centigrade ($\frac{2}{3}$° F.) in the morning until the fastigium is reached. During the fastigium the pyrexia continues, but with a daily remission and an evening rise of $\frac{1}{3}$° C. ($\frac{2}{3}$° F.) as in the normal temperature of health, until the defervescence, which frequently presents first a remittent, then an intermittent type of pyrexia, and often ends with a few days of a subnormal temperature before the normal is again attained.

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In relapsing fever the pyrexia is broken by intermissions. These, as we now know, correspond to the periods during which the spirochæte is absent from the blood. Its reappearance leads to a new accession of pyrexia on the day following this event, and the relapse may be repeated several times.

A similar condition is to be seen in true relapses of enteric fever, of which again there may be more than one. The cause of the relapse in the case of the former of these diseases is, of course, the reappearance in the circulation of the causal agent, the spirillum of Obermeyer. Of the latter a very ingenious and probable explanation has recently been suggested by Herbert Durham. This is as follows:—An ordinary attack of typhoid fever is probably due to infection by a number of different varieties of the *Bacillus typhosus*. One or more of these varieties may be only feebly represented, and perhaps multiply slowly in the infected animal. These will accordingly give rise to very slight production of their special anti-substances, though anti-substances for the other and more numerously represented varieties are formed in quantity and convalescence begins. If now from any change in the conditions in the body one of the feebly represented forms takes on an active growth there will not be sufficient of its special antibodies present to arrest it. Hence a relapse occurs until these special antibodies have been formed. The same events might be repeated with another variety after the termination of the first relapse. This theory would explain the occasional occurrence of several relapses, and the fact that they are rarely fatal. It is, however, pure hypothesis.

An intermittent fever is one in which a rapid accession is followed by a very short fastigium and rapid defervescence, after which there is a period of apyrexia to which succeeds a new accession, fastigium, and defervescence as before, and so on. These are the periodic fevers of which the tertian ague is a good example.

Hectic fever presents irregular pyrexia with a tendency to normal or subnormal morning temperatures.

The term aseptic fever is employed in contradistinction to the fevers of infective origin.

Treatment of Acne.—If suppuration has not yet occurred, the comedones should be squeezed out by means of an instrument suitable for the purpose; the part should then be washed frequently and energetically with soft soap and coarse flannel. A mixture of spirit and soap, such as the spiritus saponis alkalinus of Hebra, is useful in dissolving and softening the sebaceous matter. The skin should be disinfected by applying sulphur ointment (grs. x to ʒj), resorcin (grs. xv to ʒj of Ung. Paraffini), ichthyol, or carbolic acid in the form of ointment. When suppuration has occurred the pustules should be punctured or incised, and afterwards bathed with hot water so as to encourage bleeding, and then dressed antiseptically. The cavity may with advantage be touched with strong carbolic acid solution. Each pustule must be treated individually; the method requires perseverance, but is effectual. When the inflamed papules are of considerable size each one should be isolated by covering it with Unna's mercury-carbolic plaster mull. This should be left on for about twelve hours or more. After removal the part should be dried with cotton-wool, then washed with corrosive sublimate solution (1 in 2000) and covered with a fresh piece of plaster. In all cases of acne of the body re-infection from the clothing should be prevented by frequent changes of the garment worn next to the affected part, and washing the adjacent unaffected skin with an antiseptic wash or soap.

The practical information given in the preceding paragraph is taken from the new edition of 'Diseases of the Skin,' by Mr. Malcolm Morris, published by Cassell & Co., Ltd. This book furnishes a faithful account of modern teaching in dermatology, and is characterised by clearness of expression. The volume presents in a readable form a mass of scientific and practical observations. The author in his preface says that he has endeavoured to preserve the elementary nature of his work, and this he has not failed to do; but he also conveys to his readers such a wealth of clinical experience and diagnostic observations that the driest of the facts discussed becomes imbued with interest. To sum up, the book is remarkably useful and well arranged, and is in every way to be recommended, not only for its fulness and accuracy of information, but also for the ample facilities of reference.

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A CLINICAL LECTURE

ON

PEMPHIGUS AND ALLIED CONDITIONS.

Delivered at St. Bartholomew's Hospital, May 18th, 1903.

By J. A. ORMEROD, M.D., F.R.C.P.,

Assistant Physician to the Hospital, and Physician to the National Hospital for the Paralysed and Epileptic, Queen Square.

GENTLEMEN,—I have chosen this subject because in the hospital there happened to be two extremely rare cases, if not of pemphigus itself, at any rate of conditions allied to it, and I thought it would be a good opportunity for you to see these cases—for those of you, that is, who may not already have seen them in the wards.

The word pemphigus, to be classically correct, should be pronounced with the "i" long, as it is derived from the Greek πέμφιξ, genitive πεμφῖγος, but the shortening of the "i" has come into such common use that it is difficult to be correct without appearing pedantic. But let us try to understand what is meant by the term. You know that one of the earliest and simplest classifications of skin diseases, that adopted by Willan, was based upon what you can see on the skin. On this basis, eczema was characterised by vesicles, lichen by papules, psoriasis by scales, pemphigus by bullæ. Hence some students, I fancy, entertain the idea that any disease which gives rise to bullæ should be called pemphigus. That is incorrect, because many conditions occur, other than pemphigus, in which bullæ are formed. I take it that you know that a bulla means a bleb, or blister,—that is to say, a local upraising of the epidermis from effusion of fluid beneath it, of any size larger than that (say) of a pea. A bulla may occur in many conditions. For instance, it is one of the results of a burn, or of friction, or of injury even in normal skin, or of the inflammation of erysipelas, and of many forms

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of skin disease which are quite distinct from pemphigus. Here is a picture of erythema of the hands which has gone on to the formation of bullæ, and hence is called erythema bullosum. Here, again, is a drawing of a case of herpes in which the ordinary vesicle has enlarged so much that it is practically a bulla. Bullæ may sometimes occur in eczema, sometimes in urticaria, sometimes in lichen planus, sometimes in that puzzling disease called dermatitis herpetiformis, of which this next drawing is probably an example. There is also, as you know, a bullous form of congenital syphilide. Bullæ occur commonly enough in certain drug eruptions, particularly that due to iodide of potassium. Again, they may occur in cases of impetigo. You may remember that a case came to the skin department the other day in which a doubt arose as to whether it should be called impetigo or pemphigus, and the doubt was largely due to the presence of a large blister on the cheek. There is a peculiar disease in which bullæ occur on the hands, called cheiropompholix, which does not appear to be related to true pemphigus. Thus you see that one cannot call a disease pemphigus simply because it produces bullæ. Therefore another definition of pemphigus has been attempted—namely, that it is a disease in which, without obvious cause, and upon apparently healthy skin, an eruption arises, which from first to last consists of bullæ.

This definition certainly excludes the diseases which I have just spoken of, with the exception, perhaps, of cheiropompholix. Still, it does not appear to be quite satisfactory, and we are at present (it may be said) groping to find some type of bullous disease which runs a particular course and has a particular causation, to which we may specially apply the term pemphigus. So far as the eruption and its course are concerned, the disease known as pemphigus vulgaris may serve as such a type, though as to its causation we know very little. I show you an illustration of pemphigus vulgaris affecting the face, and another where it affected the arm. (In the description affixed to these pictures I recognise the handwriting of Sir James Paget.) In this "pemphigus vulgaris," which is no such common a disease as its name might suggest, bullæ make their appearance on normal skin, or else they may be preceded by trivial patches of erythema. These bullæ develop quickly, sometimes almost suddenly, and they run their course in this way:—Each bulla increases in size for a few days, and then it either dries up without further ado, or else an erythematous edge forms around it; its contents become at the same time opaque, and finally it dries up, leaving a scab, which clears off easily and leaves behind only a discoloured or pigmented patch; or else, probably from some unfavourable local condition, an excoriation forms, which may take some time to heal. These bullæ have not any regular distribution; they may affect any part of the skin, and may also affect the mucous membranes,—for instance, that of the mouth, pharynx, and tongue, and (it is said) even that of the intestines. One curious situation is on the conjunctiva, where they produce a condition known as "essential shrinking of the conjunctiva." Though the individual bullæ appear quickly and pass away fairly soon, and in that sense their course is acute, yet successive bullæ or successive crops of them appear, and may continue to do so for a very long time, and so the disease has been called "pemphigus chronicus." The eruption may recur even after a considerable interval of apparent health. There may or may not be itching; but in any case it is not a marked feature. There may or may not be constitutional disturbances. In some cases the temperature rises and nervous symptoms supervene, such as insomnia or even delirium, or, in severe cases, coma. There may also be diarrhœa, due, as some think, to the disease involving the intestines. Cases of pemphigus usually get well, though they may be very protracted, and relapses are not infrequent; but sometimes death occurs, either with nervous complications, such as delirium, coma, etc., or from general exhaustion, or from infection of the pemphigus blebs, by which they become erysipelatous or gangrenous. I show you a picture of a case of pemphigus which became gangrenous, and which I remember witnessing in Dr. Gee's wards when I was medical registrar.

There is a variety of the disease which is known as acute pemphigus. Here the disease is of short duration, lasting a few weeks at most, and the patient is very often acutely ill. It is apparently due to absorption of some poison from without; for Pernet and others have pointed out that it may follow a poisoned wound or a whitlow, or may affect such classes as butchers, who have to handle material which may be septic. In some cases

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micrococci have been isolated from the blebs. So it seems probable that this acute pemphigus is often a bacterial disease. But as to what the nature of the ordinary or chronic pemphigus is, or what is its exact cause, we are still quite in the dark. There are two hypotheses—one that it is dependent on some sort of bacterial infection, and the other that it is dependent in some obscure way on the nervous system. As to which or whether either of these surmises is correct, there is no definite proof. In pemphigus, and particularly the acute pemphigus, the bullæ may contain blood instead of serum. Here is an illustration of such a case described as "pemphigus hæmorrhagicus." Here is another picture of a case in which some of the bullæ were hæmorrhagic and others not. The ordinary or chronic form of pemphigus generally gets well in time, and the favourite treatment in England is by arsenic, which drug, I believe, was first introduced for this disease by Mr. Jonathan Hutchinson, who regards it almost as a specific. It does not always prove successful, however, and when it fails the best drugs to give are tonics such as quinine and iron.

In connection with acute pemphigus, I think it is worth while to refer to the subject of pemphigus occurring after vaccination. Last year, as you know, there was much vaccination done, and much was seen of rashes occurring after vaccination. Amongst them were bullous eruptions. There were two cases at this hospital—one, as you may remember, in Mark Ward, under Sir William Church. This was a man who developed a bullous rash three or four weeks after vaccination. The patient was ill with it, having a temperature of 100°—102°, and the rash continued for nearly a month before it began to subside. The board was headed by Sir William Church, "A very severe case of pemphigus following vaccination;" but he added, with due caution, "*? post hoc, ? propter hoc!*" About the same time there was a striking case in the skin department. The patient was a man who had been vaccinated three weeks before the eruption began, and when he came he was covered with bullæ, like those of typical pemphigus. Some of the bullæ were seated on normal skin; some had a red ring round them. They dried up with marvellous facility, although fresh ones kept coming out. I wanted him to become an in-patient, but he felt so well in himself that he declined to do so. There was no itching or constitutional disturbance, nor any involvement of the mucous membranes. Last year, also, some similar cases were brought under the notice of the Dermatological Society of London; one by Dr. Sequeira, of the London Hospital—a man æt. 39, in whom the eruption began three weeks after vaccination and ran its course without any rise of temperature, in very much the same way as the last case I have quoted to you. Cultures from the bullæ were made by Dr. Bulloch, but the result was negative. The verdict of the Dermatological Society was that it was obviously pemphigus, but doubt as to its connection with vaccination was expressed by some members. Dr. Freeman, of Reading, published in the 'British Journal of Dermatology' (January, 1903) notes of another case, in which the rash began three weeks after vaccination. The patient's age was 54, and he was seriously ill. He appears to have been rather a nervous subject. He had a temperature of 102°, and was very collapsed. Seven eighths of his body was covered with a rash, the bullæ varying in size from that of a threepenny piece to that of a crown piece. Some of these bullæ were slightly hæmorrhagic. In spite of the extent of the disease, however, he got well in a few weeks. You may notice the definite interval at which the eruption followed the vaccination in these cases. I think this points to some other connection than mere coincidence. Perhaps such cases constitute a special form of acute (bacterial) pemphigus which gets well without much trouble. Probably, also, we ought to reckon with the acute bacterial pemphigus that form which is known as "pemphigus neonatorum." This sometimes occurs as an epidemic amongst newly born children; indeed, it may follow the practice of a particular midwife. It is thought that this form of pemphigus is of a bacterial nature.

I pass now to consider some conditions allied to pemphigus. First I would mention a variety which is called pemphigus foliaceus. This is extremely rare, and it is probable on the whole that it is a variety of true pemphigus. It is characterised by bullæ, which are flaccid, not tensely distended, and readily burst, and are found to contain a sticky exudation. The bullæ do not heal like those of ordinary pemphigus usually do, but the exudation seems to penetrate and burrow beneath the epidermis in the neighbourhood, raising it into large

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flakes, which cover in unhealthy excoriations. The special characters of the disease are—the leaf-like flakes of separating epidermis (from which it takes its name), the incomplete healing of the lesions, the continuous spread of the disease, the frequent affections of the mucous membranes, and the result, which is generally fatal.

I shall now show you the particular cases I referred to earlier in the lecture. The first patient, a middle-aged woman, is the subject of what is sometimes called congenital pemphigus, and sometimes by the long but appropriate name "epidermolysis bullosa." Apparently it has not much to do with true pemphigus. The characters of it are comparatively simple. It is a congenital condition; sometimes it is a family disease; sometimes even it is hereditary—that is to say, passed on from parent to child. It consists in this, that the patient's skin is so peculiarly constituted that bullæ readily form on very slight irritation or injury, and these bullæ often contain blood. They usually form on the parts exposed to knocks or slight friction, such as the extensor aspects of the legs, the points of the knees or elbows, or the knuckles. The nails are often lost, and the mouth may be affected. The epidermis after a time assumes a peculiar thinned appearance, in which it looks like creased paper. The disease is not in itself dangerous to life. Although this patient is an illustration of a very rare disease, she is personally well known to many dermatologists in London. Her earliest remembrance is an attendance at the Children's Hospital, Great Ormond Street. She has been under Dr. Beevor, Dr. Colcott Fox, and Dr. Galloway, and has been twice in this hospital. In 1896 Dr. Galloway noted that the enamel of the teeth appeared very imperfect. Her teeth got very much worse afterwards, and now she has none left. In the 'British Journal of Dermatology' for 1897 you will find an account by Dr. Wallace Beatty of all the published cases of this disease, and a picture of a hand which is very much like hers. One family subject to this disease came under the observation of Dr. Wickham Legg, one of my predecessors in the skin department here—*vide* the nineteenth volume of our Hospital Reports.

The next case, which I show you by the kind permission of Sir Dyce Duckworth, under whose care the patient is, is an example of the very rare condition called "pemphigus vegetans." For the classical descriptions of this disease I refer you to a paper by Neumann in the 'Vierteljahrschrift für Dermatologie und Syphilis,' 1886, and another by Dr. Radcliffe Crocker in the seventy-second volume of the Medical and Chirurgical Society's 'Transactions,' giving all the cases up to date, and one of his own. Dr. Crocker's case is the only typical case published in England until Dr. Allen Jamieson, of Edinburgh, published another in the 'Journal of Dermatology,' August, 1902. Notes of the present (Sir Dyce's) case appear in the same journal, January, 1903. This is a most remarkable disease, and it nearly always ends fatally. Probably it has little in common with pemphigus vulgaris, save only the initial formation of bullæ. Ulceration of the mouth, or perhaps of the vulva or anus, generally marks the commencement of the disease. Upon the skin it begins with the formation of bullæ. These do not scab over and heal readily as in ordinary pemphigus, but leave a raw surface, and if that surface heals, deep pigmentation takes place; or else, and particularly in moist situations, such as the axillæ and groins, warty excrescences or "vegetations" arise from the base of the bullæ, from which comes a fœtid discharge; hence the name "pemphigus vegetans." It is worth noticing that these vegetations, especially when they arise round the anus or vulva, can be mistaken for condylomata. That is a grave mistake to make, because the disease has nothing to do with syphilis. The surface becomes widely affected, and pain and exhaustion, together with the chances of septic absorption, and the difficulty of feeding owing to the state of the mouth, all contribute towards a fatal termination. As to the causation of this disease, we do not know anything as yet. It has been supposed, as with many other diseases, that the nervous system is primarily at fault, and Dr. Jamieson, in the paper I have referred to, says that in his case there was found a degeneration of the cells of the spinal cord; but whether this was due to some toxin acting both on the skin and the spinal cord, or whether degeneration of these cells was the primary mischief, does not seem quite certain. The patient whom you now see has been a gamekeeper, and has lived a healthy country life. Until six months before he was admitted to the hospital (which was in November last) he had good health, save that he had been subject to pain in the limbs since he

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caught a chill ten years ago, and that nine years ago he injured his left hand, an injury which was followed by erysipelas. But with those exceptions he was quite well until six months before admission, at which time his lower lip became sore and excoriated, his feet began to ache, and blisters formed on them, from which came clear fluid. Then the skin became black and peeled. Of this he got well, according to his own account. But soon he had a second attack; blisters again formed on the feet, and the skin then became permanently black. The eruption spread up the legs, the scrotum became affected, and red circles appeared, followed by blisters. Three months before admission his arms and hands became affected, blisters formed there with clear contents, and subsequently the skin got dark and the limbs were swollen and painful. I invite your attention now to the following points:—The mouth and lips are sore (though they have improved since admission). There is extraordinary thickening of the epidermis on the soles and palms. The skin of the forearms and legs is inelastic and deeply pigmented. There are numerous other pigmented areas, for instance round the umbilicus. In the groins, the perinæum, and the axillæ, there are large patches of vegetations, *i. e.* of moist prominent excrescences. No bullæ are now visible, though we have seen some since he has been in the hospital. Upon the back (as Sir Dyce kindly showed to me the other day) the disease has quite recently made a fresh start. Here it is progressing, not by the formation of bullæ, but of reddish, somewhat raised papules, on which vesicles form. These vesicles very readily rupture, and then disappear, leaving a sort of umbilicated papule. The summit of these readily bleeds, and the sanious discharge from them soaks the clothing. Next the papules enlarge into flat raised areas, which spread at the periphery and coalesce with their neighbours, becoming at the same time pigmented. This exacerbation of the disease, and the rapid extension of it which is taking place over the back and over the occiput, raise very grave apprehensions for the future of the patient.

NEUBURGER reports two cases in which myopia occurred in the course of diabetes mellitus. He calls attention to the fact that myopia occurring in old age is always a suspicious symptom.—*Philad. Med. Journ.*, June 6th, 1903.

ON THE OPERATIVE TREATMENT OF FRACTURES.

Delivered at Guy's Hospital, May, 1903.

By W. ARBUTHNOT LANE, M.S.,
Surgeon to Guy's Hospital and to the Hospital for Sick Children.

(*Continued from page* 167.)

IF you examine the radiographs of a number of spiral fractures of the tibia and fibula, such as are illustrated in Figs. 7 and 8, you recognise that the displacement of the fragments is practically identical in all cases, as in the corresponding fractures of the femur. The lower fragment is displaced upwards, backwards, and outwards, while the upper is displaced forwards, downwards, and inwards. The level of the fractures of the two bones is very variable, that of the fibula being usually placed very much higher than that of the tibia. I have discussed fully the manner in which these fractures are produced, and the explanation of the displacement of fragments, in a paper published in the 'Practitioner' for March, 1903, entitled "On the Mode of Production of Fractures of the Lower Extremity by Indirect Violence." So I do not propose to deal here with the causation of these injuries, but will confine myself to their treatment.

Fractures of the tibia, whether produced by direct or indirect violence, are more likely to be accompanied with comminution of the fragments than in the case of the femur. This comminution often adds considerably to the difficulty of establishing accurate continuity. It is much more frequently present in fractures produced by direct than by indirect violence. As you see from these radiographs of fractures of the tibia and fibula resulting from direct violence, the bones are broken usually about the same level, the planes of the ends of the fragments being sometimes somewhat oblique, but frequently nearly transverse and irregular. On this account the displacement of fragments in a transverse plane is much greater in fractures resulting from direct injury, since it cannot be less than the diameter of the tibia, but there need not of necessity be any considerable alteration in the relationships of the axes of the fragments to one another.

On the other hand, in the spiral fracture the displacement in a transverse plane may be much

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less, and may not exceed half the breadth of the tibia; but the alteration in the axes of the fragments is considerable because of the rotation of the lower upon the upper.

These conditions render it more easy to replace the fragments of fractures by direct injury in apposition, but more difficult to retain them immovably on one another. In the case of the spiral fracture the difficulty of effecting accurate apposition may be very great, but once the fragments fit they can easily be secured absolutely immovably on one another by means of a screw or wire.

In operating on these fractures of the tibia and fibula it is wiser to attack the tibia in the first instance, for the reason that when the fragments have been brought accurately into apposition those of the fibula have frequently come together also. This is more commonly the case in fractures by indirect than by direct violence. Again, in the former the fracture of the fibula is placed so often in the upper part of the bone, and the displacement in a transverse plane is so small when the fragments are brought down to the same level, that even if the broken surfaces do not fit accurately the influence exerted by this displacement on the

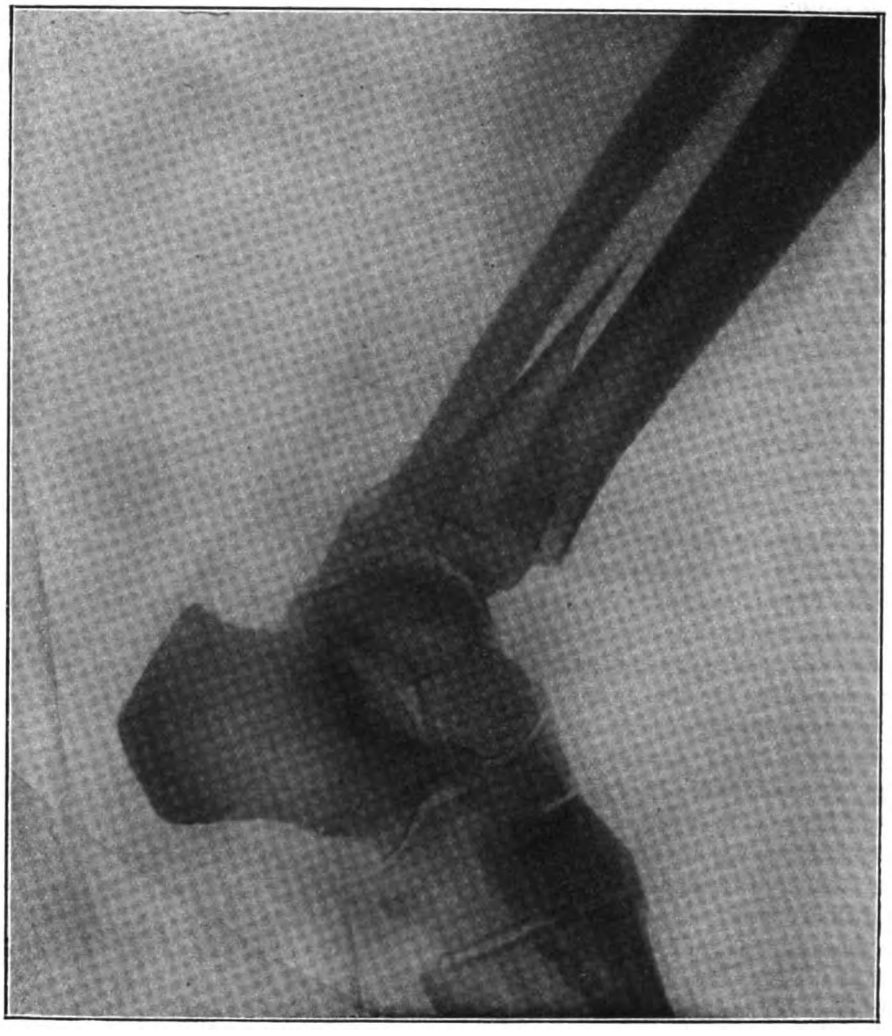

FIG. 7 represents displacement of the upper fragment downwards, forwards, and inwards; and of the lower fragment upwards, backwards, and outwards.

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external malleolus is so small as to be practically negligible.

I prefer to make the incision for the tibia about a quarter of an inch behind the anterior limit of its inner surface. It should be sufficiently long to make manipulation of the fragments easy without affording the slightest chance of the fingers or skin of the patient coming in contact with the surface of the wound.

Its length must vary with that of the fracture and with the extent of hæmorrhage, laceration of soft parts, and inflammation.

By the exercise of forcible traction on the foot, the approximation of the fragments by bone forceps, and their adaptation to one another by elevators, the fragments can be got into accurate apposition.

If the fractures be spiral or very oblique, the fragments can be held securely by Peter's forceps; but if it is transverse, or approximately so, they will

FIG. 8.—Displacement of the upper fragment of the tibia downwards, inwards, and forwards; and of the lower fragment upwards, backwards, and outwards.

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be retained by the resistance offered by the stretched and swollen ties.

In the former case a screw or wire will suffice to secure firm apposition. Such results are shown in Figs. 9 and 10.

If, however, this fracture has been produced by direct violence, and is in part or entirely transverse, a screw cannot serve any very useful purpose. In such cases I prefer to perforate each fragment transversely, and to pass a stout silver wire through each. Having twisted both ends on one side and hammered down the curves, any slack can be taken in and the loop made taut by twisting the ends of wire on the other side at the same time. In fractures by direct violence it is always necessary, or rather advisable, to expose the fibular fragments and to treat them in the same way, except that a thinner and a single wire may be used and a simple loop employed.

Fig. 9.

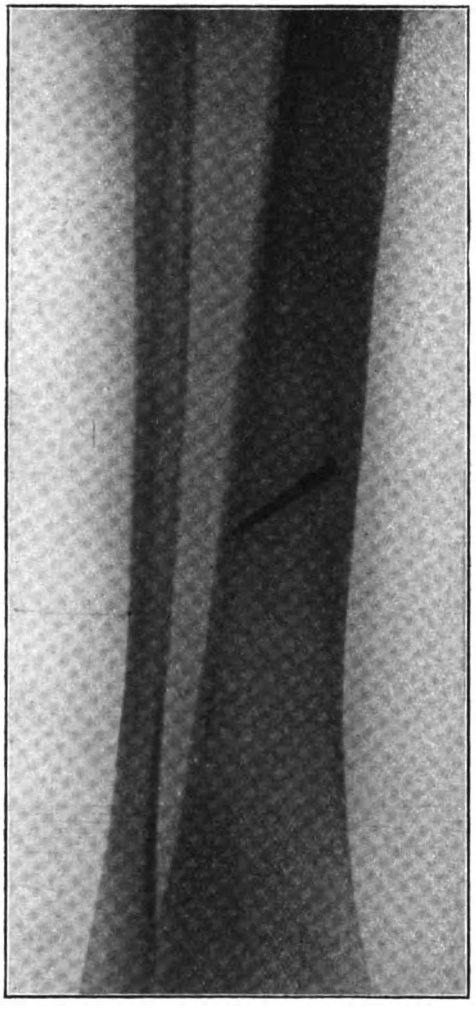
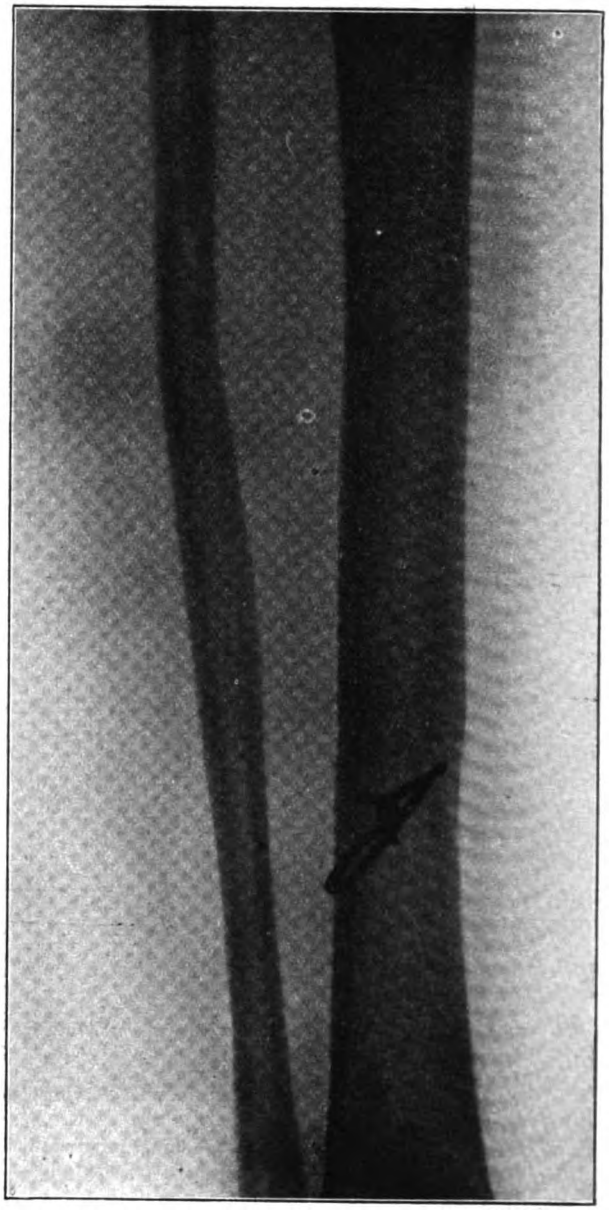


Fig. 10.



As I said before, comminution of fragments ad % largely to the difficulty of operating with perfect success on the tibia; and this applies more to the spiral fracture than to the result of direct injury,

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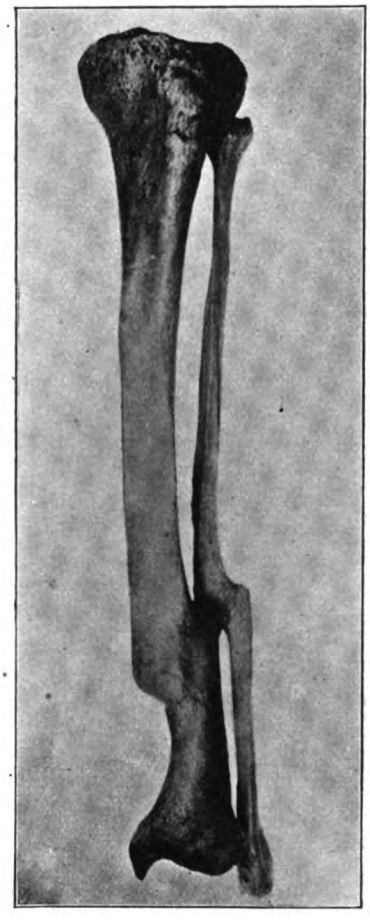
since in the latter there is almost always a portion of the end of the bone which is transverse in direction, and on which the corresponding surface can be placed, so opposing overriding. In the fracture produced by torsion there is, of course, no transversely placed ledge, and the strain exerted by the shortened ties is constant and enormous. If both terminal spikes are broken, it may be impossible to restore the bone to its normal form; but if one the seat of fracture. The length of the incision does not add in the slightest to the risk run by the patient. Figs. 11 and 12, illustrating cases of fracture of the femur and of the tibia and fibula which have been treated by means of splints, show the displacement of fragments I have described and the mechanical changes in the articular surfaces which result from the alteration in their functions, consequent on the changes in the direc-

FIG. 11.

FIG. 12.



be left intact even for a sufficient portion of its length, together with the corresponding angular interval, the small fragments may be secured to them and to each other by loops of wire.

The incision to expose the fibular fragments may be made most advantageously along the intermuscular septum between the peronei muscles and those in front of them, and, as in all these incisions, should be made so as to afford very free access to tions in which pressure was transmitted through them.

Passing on to the treatment of such fractures as result from excessive abduction of the foot, commonly classified under the heading Pott's fracture, you see from Fig. 13, which represents the ordinary form of this injury, that the lower fragment of the fibula is displaced upwards and backwards. Having obtained accurate appositi on

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of the broken surfaces, the tendency to overriding is not very great, for the reason that there is not the same shortening up of ties as there is in fracture of the bones of the leg higher up.

Consequently, in this form, after the fracture has been exposed and the ends approximated and perforated by a drill, a wire loop of moderate thickness is usually sufficient to hold the parts securely. If there is any doubt in your mind you can supplement the perforating wire by one or two loops encircling the fragments above and below it. If the inner malleolus is broken and displaced it should be exposed by a vertical incision, through which the fragments can be drilled and connected accurately by a loop of wire.

In Pott's fractures the limb should be kept perfectly at rest for a period varying between a month and five weeks. At the end of this time the surgeon may commence movement of the part, knowing that if he has been able to effect perfect restoration in the form of the broken bone or bones he will have none of that trouble which is almost always present for a long time, and frequently permanently in cases treated in the ordinary way—I mean by measures other than operative. I know no operation that is more com-

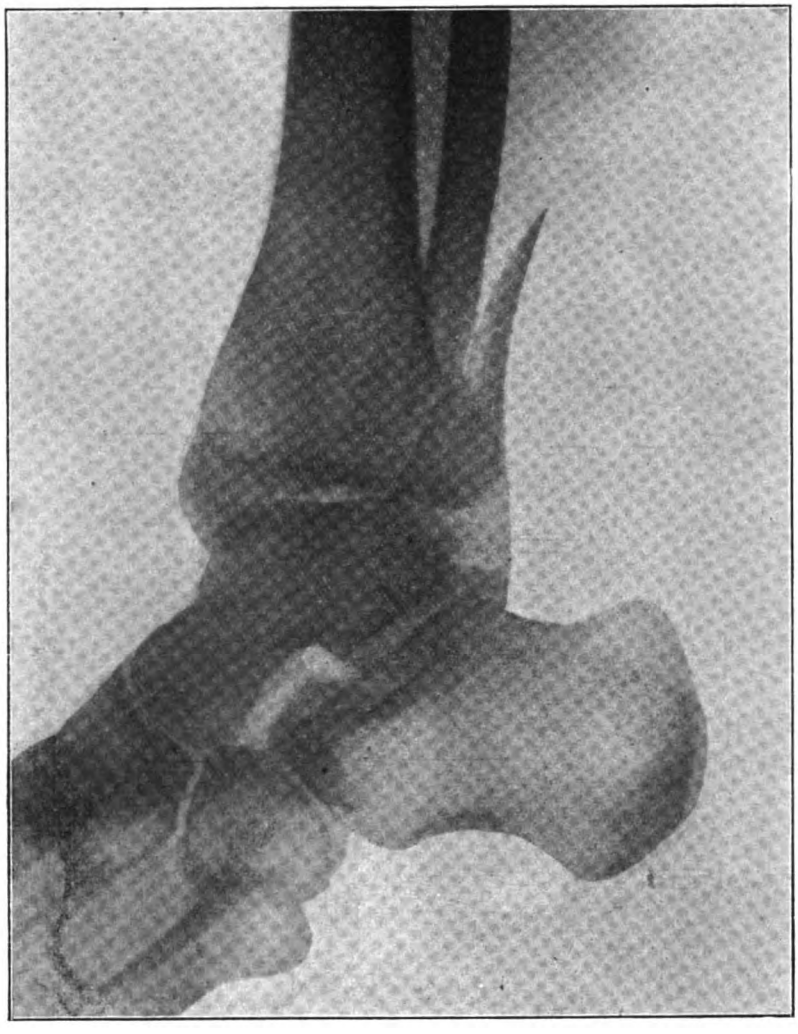

FIG 13.—Ordinary type of Pott's fracture seen laterally.

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pletely satisfactory in its results than that performed for Pott's fracture, a condition which under ordinary treatment occasionally disables the patient permanently. Besides, the risks of performing it must be very small indeed if ordinary precautions are taken.

If the surgeon has not succeeded in wiring fractures of the patella without killing or permanently disabling many of his patients, he had better bring himself to believe that the results of the generally accepted methods of treatment are excellent, and that any statements to the contrary are exaggerated or imaginary, and leave operations on recent fractures of the long bones of the leg alone, since they may test the methods and skill of the operator to the utmost.

In performing these operations, not only must you not touch the interior of the wound with your hands nor permit the patient's skin to do so either, but you must never let any portion of an instrument which has been in contact with your skin or with that of the patient touch the raw surface. All swabs must be held in forceps and applied to the wound in that manner. They should not be handled in any way previous to being used.

After an instrument has been used for a length of time, or forcibly, it should be re-boiled or placed in a germicidal solution.

I have occasionally seen beginners employ the handle of a knife or dissecting forceps to separate adherent parts, or to displace some structure, instead of using the particular instrument made for that purpose. I need not remind you that this must not be done on any pretext whatever.

The introduction of the hand into the wound in most operations, besides being dangerous, is an unnecessary and clumsy expedient. It may be resorted to in the case of the general peritoneal cavity, where only a very moderate amount of cleanliness is requisite to obtain a good result, the peritoneum possessing powers of dealing with organisms very greatly in excess of those existing elsewhere; or in such operations as nephrorrhaphy, where it is wisely safeguarded by drainage.

[We are indebted for several of the blocks illustrating this lecture to the courtesy of the Editor of the 'Practitioner.']

(*To be continued.*)

A CLINICAL LECTURE
ON
DIASTOLIC MURMURS.

Delivered at the Brompton Hospital, February 25th, 1903.

By H. BATTY SHAW, M.D.,
Assistant Physician to University College Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton.

In the study of valvular disease of the heart, it is most important that the observer should be conversant with the probable source of the various murmurs that may be heard in various regions of the cardiac area. No murmurs, probably, are more difficult of recognition and of interpretation than diastolic murmurs, and the explanation of this difficulty is to be found partly in the occasional very faint character of the murmurs, partly in the variation produced by change of posture or by exertion, and partly in the limitation of our knowledge with respect to cardiac physics.

Diastolic murmurs may occupy the latter part of diastole, and are then known as presystolic murmurs; they may, however, occupy the whole diastolic period or be confined to the early part of diastole or to the middle portion.

Diastolic murmurs are most commonly heard in one of two areas, though occasionally they may be heard at both, viz. at or near the apex of the heart and at or near the third left intercostal space of the left side, near to, or but little removed from, the left edge of the sternum. Diastolic murmurs may, however, be heard at other parts of the chest wall, viz. at the second right intercostal space near the sternum, over the body of the sternum itself, or at the xiphisternum.

A. *Diastolic Murmurs heard at or near the Apex of the Heart.*

1. *Mitral Stenosis.*—Post-mortem observation shows that cases of pure mitral stenosis are comparatively rare, as regurgitation is so often demonstrable as well. Clinically, however, a murmur indicative of stenosis is often the only one heard. It may be presystolic, early or mid-diastolic, or it may occupy the whole of diastole. It is a very localised murmur, and is confined to the area of maximum thrust of the heart or just a little

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internal to this point, though occasionally a mitral stenotic murmur is heard over a wider area than the accompanying regurgitant murmur. As a rule it is not conducted in any direction, though such a murmur has been heard at the inner border of the left scapula. Often it is heard only when the patient is upright, or less often it may be present in recumbency only; in other cases it is present whatever attitude the patient assumes. Frequently the murmur is absent for intervals of days, and returns later, so that auscultation in these intervals may fail to detect the most reliable sign of mitral stenosis. The presystolic murmur is frequently followed by a sharp first sound into which it runs with crescendo intensity; so similar is this altered first sound to the second sound that the unwary observer may interpret the stenotic murmur as regurgitant and the sharpened first sound as the second sound. Very frequently, according to the stage of the valvular disease, the true second sound is inaudible at the apex. In addition, the pulmonary second sound heard at the base of the heart may be accentuated, and reduplication may be heard in the same region. Finally, a diastolic murmur due to mitral stenosis may in the same person, at different times and under apparently the same conditions of posture and exercise, occupy different parts of the diastolic period; and in some cases auscultation at one part of the area in which the murmur is heard may reveal the fact that it occupies one particular part of diastole, whereas at another part of the generally limited area in which mitral stenotic murmurs are heard, it is observed to occur in a quite different part of diastole. Mitral stenotic murmurs are usually better marked when the patient is sitting or standing, mitral regurgitant murmurs when the patient is recumbent. As Phear and other observers have shown, an apical presystolic or mid-diastolic murmur may be heard independent of mitral endocarditis or sclerosis, and depends upon the production of a fluid vein in the left half of the heart, consequent upon dilatation of the left auricle or left ventricle, or increase in the circumference of the mitral aperture, or shortening of the chordæ tendineæ.

2. *Aortic Regurgitation.*—The maximum murmur of aortic regurgitation may be quite localised to the apex of the heart, and may occur in early diastole, mid-diastole, the whole period of diastole, or only in the presystolic period, though in the latter case it has not the character of the crescendo presystolic murmur of mitral stenosis. An aortic regurgitant murmur, heard at its maximum at the base, is frequently heard at the apex as well. It is very common to hear this murmur localised to the apex in the erect posture, and yet when the patient is recumbent the murmur can no longer, or but faintly, be heard at the apex, and is heard at its maximum further towards the base of the heart. The similarity of this murmur, when heard at the apex only, to that produced by mitral stenosis, is greatly intensified when, at the apex, a systolic murmur is heard as well; such cases are frequently diagnosed as cases of double mitral disease when met with amongst out-patients, and yet, on admission to the wards, they prove to be cases of aortic regurgitation with dilatation of the mitral valve from enlargement of the left ventricle, or regurgitation through the mitral valve from associated endocarditis of that orifice.

To confirm the diagnosis of aortic disease, observations must be made for Corrigan's pulse, for capillary flush, and for the production of a diastolic murmur in the femoral artery on pressure with the stethoscope (the vascular diastolic murmur of Duroziez).

When diastolic murmurs are heard at the apex in two different periods of diastole, care must be taken to remember that such phenomena may be due to decrescendo and crescendo changes of a mitral stenotic murmur; but it must also be remembered that mitral stenosis not infrequently occurs associated with aortic regurgitation. The aortic origin of a regurgitant murmur may be more readily recognised when the patient is recumbent than when erect, though the *intensity* of the murmur is not so much influenced by change of posture as is the intensity of the murmur of mitral stenosis.

3. *Flint's Murmur.*—This is a murmur heard in diastole, at or near the apex of the heart, in cases of aortic regurgitation. By some observers it is considered to be the aortic regurgitant murmur which, though better heard at the base, is also audible at the apex, and is lost in that area of the chest wall intervening between these two sites. This murmur usually occurs in mid-diastolic or in the presystolic period, so that there is much difficulty in differentiating it, on the one hand, from an

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aortic regurgitant murmur, and on the other, though to a much less extent, from a mitral stenotic murmur; but an aortic regurgitant murmur heard at the apex and occupying the latter part of diastole does not run in a crescendo manner into the first sound, and mitral stenosis reveals, usually, such signs as accentuation of the first and second sounds and reduplication of the latter, which are not present when a Flint's murmur is heard. Some observers affirm that in all cases of aortic regurgitation in which a presystolic murmur is heard at the apex of the heart, having a crescendo character, mitral stenosis is found, post mortem, as well as aortic regurgitation; but Thayer has recently found that Flint's murmur was heard in a little more than half of the cases of aortic insufficiency, uncomplicated, as shown by post-mortem examination, by mitral stenosis.

Unanimity of opinion on the possibility of the occurrence of a Flint's murmur can scarcely ever be attained: firstly, because all observers are not agreed that it is possible for an aortic murmur heard at the base of the heart and at the apex to be lost in the area of the chest wall intervening between these points; and secondly, because mitral stenosis may produce a murmur in mid-diastole only, and may be unaccompanied by a sharp first sound or accentuation and reduplication of the second sounds at the base of the heart.

4. *Functional Diastolic and Presystolic Murmurs other than Flint's Murmur.*—These murmurs may occur in early diastole, mid-diastole, or in the presystolic period, or, theoretically, even throughout the whole of diastole. Unlike Flint's murmur, they occur independently of aortic regurgitation. These murmurs, as already mentioned, are met with in those cases in which there is an alteration of the normal relationship existing between the left auricle, the mitral ring, and the left ventricle. You will hear them in cases in which adherent pericardium is associated with dilatation of the left auricle or left ventricle; in cases of chronic renal disease, or arterio-sclerosis accompanied by dilatation of the left ventricle and mitral ring; and also in cases in which dilatation of the left ventricle is dependent upon profound anæmia or the action of the poisons of acute infectious disease. In some cases the chordæ tendineæ of the mitral curtains are thickened and shortened, though the valves themselves are quite normal.

B. *Diastolic Murmurs heard in the Vicinity of the Third Left Intercostal Space near to or but little removed from the Sternum.*

1. *Aortic Regurgitation.*—The aortic orifice is placed near the sternum at its junction with the third left costal cartilage, and according to different observers the larger number (90—95 per cent.) of cases of aortic regurgitation are revealed by diastolic murmurs heard loudest in the left intercostal spaces near the sternum; in some cases the maximum murmur is heard in the second left intercostal space, or even in the third or fourth left intercostal spaces near the sternum, or over the middle of the length of the body of the sternum. The line of conduction may be towards the xiphisternum or towards the apex of the heart; in some cases the murmur appears to lose itself along the line of conduction towards the apex, appearing again at the apex-beat of the heart. The aortic diastolic murmur is generally an early diastolic one; often it occupies the greater part of diastole; in either case it is usually louder at its beginning than at its termination, sometimes it is louder at the mid-diastolic period, and it may be louder towards the end. This murmur may, however, be mid-diastolic only, or even presystolic. Considering the gravity of the lesion producing this murmur, it is most important that it should be recognised; such a murmur is frequently misinterpreted by the beginner; it is often so loud that it is considered to be a systolic murmur. As a rule, it is not of much use to depend upon the character of a murmur as a means of diagnosis of the lesion producing it, but there are occasions, as in this case and in the case of the murmur of mitral stenosis, when the character of the sound is of great importance; the familiar crescendo murmur of mitral stenosis is often relied upon by the beginner, who is unable, as yet, to accurately estimate the time of murmurs; such reliance is, however, not to be recommended. An effort should be made quite early in the examination of cardiac cases to accurately fix the period of the cardiac cycle in which the murmur or murmurs appear. An aortic regurgitant murmur is often met with which is quite musical in character, and it may be audible without the use of the stethoscope at some distance from the chest. Frequently, such a murmur is thought to be systolic in time, on the mere assumption that all

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such musical murmurs are more likely to be systolic; an appeal to the time relationship of such a murmur to the thrust of the heart would readily correct this mistake. It is commonly taught that aortic regurgitant murmurs may be heard in the vessels of the neck, but however lightly the stethoscope is placed on the carotid vessels, there is always the risk with beginners that excessive pressure may produce a fluid vein and an accompanying local vascular diastolic murmur which is quite independent of the murmur produced at the aortic orifice; and it is further not very probable that a diastolic aortic murmur should be heard in a direction directly opposite to that in which the regurgitant stream is flowing. In some cases, however, a regurgitant aortic murmur is so marked that it can be heard in many directions both at the front and back of the chest. Cases frequently come under observation in which a diastolic murmur is heard in the precordial area, and, for various reasons, hesitation is experienced in diagnosing aortic regurgitation; and we are accustomed in such cases, when no evidence of displacement of the apex-beat is forthcoming, nor other general signs and symptoms of aortic disease are manifested, to rely upon the three following signs, the existence of which is said to be confirmatory of leakage through the aortic aperture, viz. the water-hammer or Corrigan pulse, capillary pulsation, and the vascular diastolic murmur of Duroziez. In the case of the first of these three signs, the patient's wrist is grasped by the examiner's hand in such a way that the anterior part of the wrist is closely apposed to the palm of the observer's hand; with the patient's arm dependent, an estimate is made of the character of the impulse thus felt in the vessels at the wrist; the arm is then raised, and if the impulse now has a more brief, shock-like character, the observer registers such as a water-hammer pulse; but many individuals when examined thus will show an alteration in the pulse shock; indeed, most people will do so quite independent of aortic regurgitation, for with the arm raised gravity comes into play and reduces the duration and force of the radial pulse. The capillary pulsation revealed by rubbing the skin of the forehead is also met with independent of aortic regurgitation; for example, many healthy people after a full meal will show this sign. As has been justly said, capillary pulsation should be looked upon rather as a measure of arterial tension and of the force of contraction of the left ventricle, which may be reduced by so many conditions other than aortic regurgitation. The vascular diastolic murmur of Duroziez, when present, is, however, of great value, and only a very few observers have met with it independent of aortic regurgitation; it is conceivable that such a murmur could occur in that period of the cardiac cycle just preceding the closure of the aortic valves, but so far as personal experience is concerned, no case has been met with in which a Duroziez murmur has been heard, and, post mortem, aortic leakage has been excluded. The great drawback to the application of this valuable sign is that very frequently it cannot be elicited in cases of aortic regurgitation, at least in those vessels which can be clinically examined.

2. *The Pulmonary Regurgitant Murmur of High Tension.*—To those of you who are beginners in the study of clinical medicine, the discussion of a murmur such as this may be quite unheard of; it is considered a topic to be shunned by students, and rightly so, for you have many difficulties to surmount in the mastery of murmurs which have an obvious pathological explanation, without being troubled with such a murmur, the genesis of which is so imperfectly understood. But now that so much more attention is being paid to the auscultation of the chest wall to the left of the upper sternum when investigating cases of supposed aortic disease, difficulties have arisen as to the interpretation of an early diastolic murmur heard at its maximum in the second, third, or even fourth left intercostal spaces near the sternum, or in the third left intercostal space midway between the left edge of the sternum and the left nipple line. Such a murmur is fairly frequently met with in cases of mitral stenosis, and two such cases will be shown to you presently. It will be instructive to hear briefly the clinical and pathological observations made upon such a case formerly under treatment at University College Hospital.

A girl æt. 14 years was admitted with joint pains and pain over the precordium; the apex-beat was felt in the fifth left intercostal space internal to the nipple line; the ordinary signs of double mitral disease were present; the lungs were congested, and there were signs of failure of the right heart. In the second left intercostal space near the sternum an early diastolic murmur was present, having its

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maximum at this spot; it was also heard in the third and fourth left intercostal spaces near the sternum. At the second right intercostal space near the sternum no trace of this murmur could be heard; capillary pulsation was present, but it was doubtful whether the radial pulse was of the Corrigan type, and the murmur described by Duroziez could not be elicited. So marked, so variable in intensity, so superficial was this murmur that, considering the elevation of the temperature, the precordial pain, and the pain in the joints, a diagnosis was made of pericarditis, and the sudden disappearance of the murmur shortly before death tended to confirm this view. Another opinion was that it was a regurgitant aortic murmur. Death occurred a week after the discovery of the murmur. There was a button-hole mitral valve; on the tricuspid valve, which admitted four fingers easily, recent vegetations were found. The aortic and pulmonary valves were free from disease, and appeared quite competent, and, finally, there was not a trace of pericarditis.

Graham Steell, Barié, Gouraud, Stokes, Gibson, and more recently Bryant, have published reports on similar cases, Bryant giving an account of sixteen such cases, nine of which were examined post mortem. The suggested explanation for the genesis of this murmur is that the pulmonary artery, subjected to the strain induced by the hampered pulmonary circulation, dilates, and with it the pulmonary ring, so that a temporary functional regurgitation takes place. A similar explanation has been offered for a temporary aortic regurgitant murmur occurring independently of disease of the aortic valves. You will meet with the so-called pulmonary regurgitant murmur of high tension, but you must be warned and advised before you diagnose it to *carefully exclude the existence of aortic regurgitation*, or you will be led into the grave error of overlooking this serious lesion, and add your experiences to those who have learnt that there is much danger of error when dealing with a diastolic murmur occurring in what has been called the "area of romance"—viz. in that part of the chest near the pulmonary cartilage.

3. *The Late Systolic and Diastolic Pulmonary Murmur of Congenital Heart Disease.*—The "blue disease" of children and adolescents is quite familiar to you, and you are accustomed to associate cyanosis, clubbing of the fingers, and a systolic murmur heard at the pulmonary cartilage and propagated towards the left clavicle, with stenosis of the pulmonary artery, imperfect septum ventriculorum, and tricuspid regurgitation. A case will, however, be shown to you presently of a boy æt. 6 years. He is but slightly blue, and has no clubbing of the fingers; but all his life, even at birth, the precordium has been noticed to be the seat of wide-spread pulsation; the heart is somewhat enlarged, and there is a very distinct thrill to be felt to the left of the sternum in its upper part. This thrill is often purely systolic, but occasionally there is some thrill to be felt which occurs a little later than the thrust of the heart. On auscultation a very loud, harsh systolic murmur is heard over the same area in which the thrill is felt. The child on one occasion was admitted to the wards with the diagnosis of pulmonary stenosis, the evidence being the systolic thrill and murmur in the region indicated. Examination later, however, revealed the fact that part of the thrill and murmur occurred in diastole, and the remainder occupied the latter part of systole. Gibson has described somewhat similar cases, and has confirmed by post-mortem examination in one case the diagnosis made during life—namely, of patent ductus arteriosus. Such cases may show patency of the ductus arteriosus, combined with dilatation of this vessel and with other congenital lesions.

c. *Other Areas in which Diastolic Murmurs are heard.*

In cases of double aortic disease a "to-and-fro" murmur occurring in systole and diastole near the second right cartilage is a familiar sign, and the regurgitation is confirmed by finding the maximum diastolic murmur either at the second right intercostal space or in the various situations already referred to. The difficulty in excluding pericardial friction-sounds in such cases is well known to you, and it only remains to remind you of the value of studying in such cases the line or lines of conduction of such sounds and the superficial creaking character of the pericardial friction-sound, and to remind you how much more pericardial friction-sounds are synchronous with the heart's movements than with the heart's sounds.

Aortic regurgitant murmurs are sometimes audible at their maximum at the xiphisternum or at the left margin of the lower part of the sternum. Very

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rarely a tricuspid stenotic murmur may be heard at the left of the sternum near its junction with the fifth and sixth costal cartilages. Such a condition is comparatively rare, and would be readily differentiated from an aortic regurgitant murmur by the consideration of the signs and symptoms peculiar to these two affections. Cases, too, are met with, but very occasionally, in which a diastolic murmur is heard over the left margin of the sternum, due to pulmonary incompetence dependent upon organic disease of the pulmonary valves. Diastolic murmurs due to aortic regurgitation, when of sufficient intensity or of a musical character, may be heard in most parts of the chest, back and front.

In choosing the subject of diastolic murmurs for the lecture this afternoon, I am fully aware of the difficulty of the subject, and would ask you to remember that here, as in so many other conditions met with in chest disease, it is the stethoscope which so often sounds the necessary alarm and calls our attention to the need for a more careful and extended examination of the chest; but it is this extended consideration alone that will help you to a true estimate of the nature and degree of the valvular defect and of the powers of compensation still possible to a heart the seat of disease. The time allotted to this lecture is too brief, however, to discuss the further examination of the heart, but enough has been said to show the importance of a careful auscultatory examination of murmurs diastolic in rhythm; and, in conclusion, I would recommend to you the plan I have adopted in this lecture of bearing in mind all the possible known causes of a diastolic murmur heard in different regions of the chest.

We have received from Messrs. Burroughs Wellcome and Co. a specimen of "Soloid" Romanowsky Stain (Leishman's powder). One "Soloid" product is to be dissolved in 10 c.c. of pure methyl alcohol. A few drops of the solution should be run on to the blood-film, and allowed to remain four or five minutes. Then double the volume of distilled water is to be dropped on to the film. This will bring out the red tint in the corpuscles and blue colour in the nuclei. After another two minutes' staining the film may be washed in distilled water, dried in air, and mounted in xylol balsam.

Exploratory Laparotomy.—At the present day there is, in diseases of the abdomen generally, rather too great a tendency to the employment of an exploratory laparotomy to the exclusion of other methods of diagnosis. The exploration should only be employed as a diagnostic measure when other means of diagnosis fail. But whenever the symptoms point to some condition that is remediable, or when there is doubt as to the applicability of several methods of treatment, an exact diagnosis is imperative for the employment of the proper remedial measures, and an exploratory laparotomy should be done. No exploration should be practised, however, even in the cases we have just mentioned, without the surgeon being prepared to go on to carry out whatever method of treatment seems most suitable. It has happened somewhat unfortunately that large inoperable tumours have been met with when the abdomen has been opened, for which obviously nothing in the way of removal could be done; and, notwithstanding this, improvement or even cure has followed after the abdomen has been shut up. This has undoubtedly given a fillip to exploratory laparotomy even in hopeless cases, under the impression that, as a result of the laparotomy, the tumour might disappear. It is hardly conceivable that such tumours are malignant. Greig Smith has pointed out that in several of the cases upon which he operated the tumours were merely inflammatory; and to cut into an abdomen containing a typical cancerous tumour on the chance of the laparotomy leading to the disappearance of the tumour is a practice that cannot be too strongly deprecated.

The instructive paragraph quoted above is taken from Section I of Part VI of 'A Manual of Surgical Treatment,' by Mr. Watson Cheyne and Mr. Burghard. It has not been found possible to complete this manual in six volumes, so the reasonable plan has been followed of dividing Part VI into two volumes. As the authors point out, to have attempted to include the whole of the remaining subjects in one volume would have entailed the condensation of the matter and the abandonment of the full discussion of the treatment of the various diseases, which is the leading motive of the work. Section I of Part VI deals with the surgical affections of the tongue and floor of the mouth, the pharynx, neck, œsophagus, stomach, and intestines. The volume is in every way entitled to be ranked as high as those which have been already published (Longmans & Co.), and is remarkable for its sustained excellence.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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DISAPPOINTMENTS.

By J. BLAND-SUTTON, F.R.C.S.,

Surgeon to the Chelsea Hospital for Women; Assistant Surgeon, Middlesex Hospital.

"THE best laid schemes o' mice an' men
Gang aft agley."

There is nothing in surgery which illustrates this trite phrase so forcibly as the remote results of operations on the female pelvic generative organs when carried out with the object of enabling women to become successful mothers.

In this short communication I propose to furnish the details of three cases recently under my care, illustrating the unreckoned probabilities which may mar the best efforts of the surgeon,

"An' leave us nought but grief and pain
For promised joy."

CASE 1.—In the autumn of 1900 a woman æt. 26 years was placed under my care in the Middlesex Hospital with the following story:—She had been married eighteen months, and in this short period had conceived and miscarried three times. On admission to the hospital she had conceived for the fourth time, and had advanced beyond the second month. The doctor who had attended her at the third miscarriage had detected a small movable body in the pelvis, and advised her, should she again become pregnant, to seek surgical help. On the strength of this recommendation she came to me for advice, and I had no difficulty in recognising an ovarian cyst the size of my fist low in the pelvis, incarcerated by a gravid uterus. The method of procedure in such conditions is now well established—namely, removal of the tumour; and I preferred to deal with it by the abdominal route. The operation presented no difficulty; the patient quickly recovered, and in due course brought forth a healthy living child at full term. In this respect the case is one abso-

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lutely without novelty, but it is of interest in showing that an ovarian tumour the size of a fist incarcerated in the pelvis by a gravid uterus can so interfere with the pregnancy as to lead to three miscarriages in the course of fifteen months. In some lectures which I published in the 'Lancet,' 1901, this case was briefly detailed to illustrate the malign influence which even a small ovarian cyst may exert on pregnancy; or, to state it in the form of an aphorism, *ovarian tumours are, as a rule, inimical to successful pregnancy.* Certainly this applies with double force when the ovarian cyst is so situated in the pelvis that it becomes incarcerated by the enlarging uterus.

To continue the history of the case :—The child throve well, and attained the age of sixteen months. One evening whilst left in front of a fire it managed to upset a saucepan of boiling water, and received a severe scalding, from which, in spite of the assiduous attentions of the resident officer and nurses at a neighbouring hospital, it succumbed in a few hours.

Case 2.—In December, 1900, Dr. Louis Beer placed under my care in the Chelsea Hospital for Women a patient æt. 33 years, on account of a tumour in the right iliac fossa. This woman had had three miscarriages. In each instance the pregnancy had advanced to the third month before expulsion occurred, and, as she was extremely anxious to possess a living child, Dr. Beer was particularly careful when the patient became pregnant for the fourth time to avoid anything that would militate against a successful issue; so that when the pregnancy reached the third month he kept her in bed. About the time of quickening the patient complained of pain, and a rounded knob of the size of a tennis ball appeared in the right iliac fossa. It was difficult to decide whether this lump occupied the abdominal wall or was intra-peritoneal. At the operation we found a sessile fibroid growing from the anterior wall of the uterus, adherent to the bladder and omentum. The tumour had so dragged on the bladder that this viscus extended to the right side of the pelvis, and probably accounted for the pain and distress which caused the patient to seek medical advice.

Without any particular difficulty I enucleated the tumour. It exhibited the mahogany tint peculiar to fibroids when they complicate pregnancy. The uterus was gently surveyed with the finger, but nothing interfered with the smoothness of its contour.

The operation immediately relieved the patient of pain. She convalesced in the quick and satisfactory manner which is such an astonishing feature of surgical operations performed on pregnant women, went to full term, and had a living child. Dr. Beer noticed during the course of the labour that some small lumps, which he suspected to be fibroids, had appeared on the anterior surface of the uterus.

It was natural that all concerned should congratulate themselves on the apparently successful results of their efforts; but the happiness soon vanished, for the child could not retain or even swallow food, and Dr. Beer came to the conclusion that the baby had an imperforate pharynx. Together with the aid of chloroform, the diagnosis was established. The baby slowly starved, and died about six days after its birth. It seemed an obvious thing to comfort, or, at any rate, to console the mother with the assurance that, though she had lost her baby, events had demonstrated her womb to be capable of retaining a living baby, and that the outlook was distinctly encouraging. During 1902 and the beginning of 1903 there were two miscarriages. After the second miscarriage metrorrhagia became very marked, and in March, 1903, the losses of blood were so frequent and abundant as to demand some radical treatment. Our knowledge of the patient's history and the size of the uterus led us to suspect a submucous fibroid, although we could not altogether exclude deciduoma. The patient, however, insisted that, as she had suffered so much, and as we were sure she had some fibroids in the uterus, she would not submit to any operation that did not aim at being radical and restore her to health. It was explained to her that this could only be attained by removal of the womb and the obliteration of all hopes of maternity.

Hysterectomy was performed in April, 1903. The uterus contained several small fibroids in its walls, and a peculiar sessile body at the fundus involving the endometrium. Macroscopically we could not decide its nature, but even in the laboratory we cannot satisfy ourselves whether it is a degenerate fibroid or a spindle-celled sarcoma. However, to-day this long suffering woman appears

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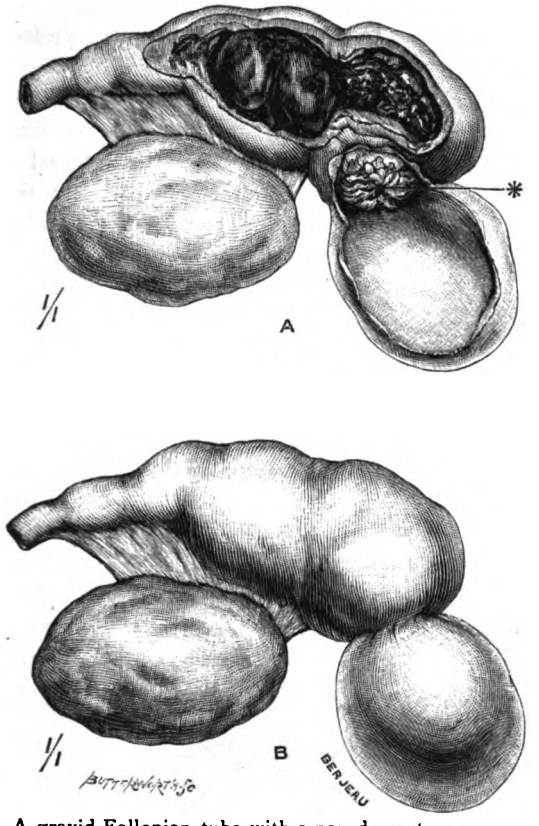
in excellent health, and though complaining of indefinite pains, and chastened by disappointed hopes of successful maternity, is nevertheless happy.



A gravid Fallopian tube with a pseudo-cyst or capsule, formed around blood which trickled or dripped through the cœlomic ostium, due to a retained "mole." B shows the capsule entire, A in section, so as to display the relation of the neck of the capsule to the fimbriæ *.

CASE 3.—In 1901 my advice was sought in regard to a young lady who complained of pelvic pain and discomfort; as she had been married some three years and remained sterile, it was thought that the trouble might be due to some undetected lesion of the internal pelvic organs. A careful examination made it clear that there were no gross lesions, and the husband cleared up matters somewhat by stating that he had taken steps from the very outset of marriage to avoid conception. We had a conversation on the subject, and came to the conclusion that the patient would be happier if she were involved with maternal cares and duties.

About a year later I was urgently asked to see this patient, for she had conceived, miscarried, and now suffered from severe metrorrhagia, believed to be due to a piece of retained placenta; and her doctor wished to dilate and curette the uterus. Before consenting to the curettage the patient's friends wished my opinion.

The history of the case caused me to be very doubtful as to the miscarriage, and on examining the patient I came decidedly to the opinion that she had a tubal pregnancy, and the free bleeding from the uterus was due to blood making its way from the tube through the uterine ostium into the cavity of the uterus, as we know often happens in cases of "incomplete tubal abortion."

The diagnosis of tubal pregnancy was established by an examination under ether, and to the satisfaction of the doctor in charge of her. The patient was admitted into the Chelsea Hospital for Women, and in March, 1902, I opened the abdomen and found the right tube occupied by an old tubal mole. At the cœlomic ostium of the tube we found an ovoid body completely enclosing the fimbriæ (see Fig.). This tube and ovary were removed, and the left tube being occluded, adherent, and dilated with blood, we deemed it wiser to remove that also.

The oval body embracing the cœlomic ostium of the right tube is an excellent example of the "peritubal hæmatocele." These capsules are particularly liable to form in cases where blood (or sterile inflammatory exudation) slowly trickles or drips from the ostium of the tube. The effused fluid becomes surrounded by a lowly organised capsule, so as to appear like an ovoid bulb (or amphora) containing blood, and its neck embracing the cœlomic ostium of the tube.

The peritubal capsule in this particular case prevented blood leaking into the pelvis, and as the fimbriated end of the left tube was completely occluded, it makes the inference probable that the vaginal fluxes were really due to blood effused into the Fallopian tubes escaping into the uterus and causing a *spurious* metrorrhagia. The patient's convalescence was uneventful, and she remains in good physical health, but yearns after the indefinite, and feels that there is something unattainable, but knows not what.

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ON THE OPERATIVE TREATMENT OF FRACTURES.

Delivered at Guy's Hospital, May, 1903.

By W. ARBUTHNOT LANE, M.S.,
Surgeon to Guy's Hospital and to the Hospital for Sick Children.

Continued from p. 187.

We will now pass on to the operative treatment of fractures of the upper extremity, and I will call your attention first to those of the clavicle. You will come across cases in which you will find yourself unable to restore this bone to anything approaching its normal form. This difficulty may be aggravated by comminution of the fragments. When it is judged necessary to operate on these cases, continuity is readily and securely established if the fragments are unbroken and entire; but if there be separate pieces which must form intermediaries between the main fragments, great difficulty may be experienced. If, as is so often the case, you are not called on to operate till after an interval of a few weeks have elapsed since the receipt of the injury, you will find that to effect the perfect restoration of the form of the bone will make a considerable demand on your skill and patience. This, however, applies to all operations for fractures of long bones undertaken after an interval of time, but the risks are increased here by the presence of large vessels and nerves in the immediate vicinity of the fracture.

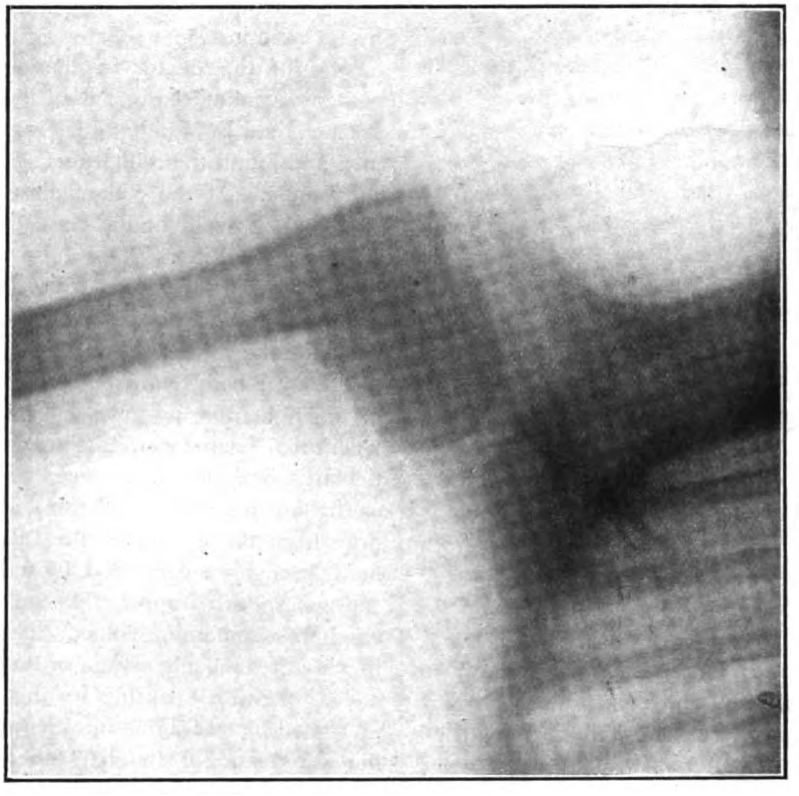

Fig. 14.—Fracture through the upper epiphysial line of the humerus with much displacement of the epiphysis. A portion of the shaft has been broken off and is attached to the epiphysis.

In this operation it is necessary that you should so arrange the twist of wire that it shall not lie immediately beneath the skin; otherwise it is sure to worry the patient at a later date and necessitate its removal.

You may possibly come across cases of fracture through the upper epiphysial line of the humerus in which you will find yourself perfectly unable to

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restore the bone to its normal form. The upper end of the shaft is displaced upwards and forwards, and projects often well above the head of the bone. Fig. 14 represents the usual displacement.

In a large proportion of these fractures a portion of the shaft remains attached to the epiphysis. It

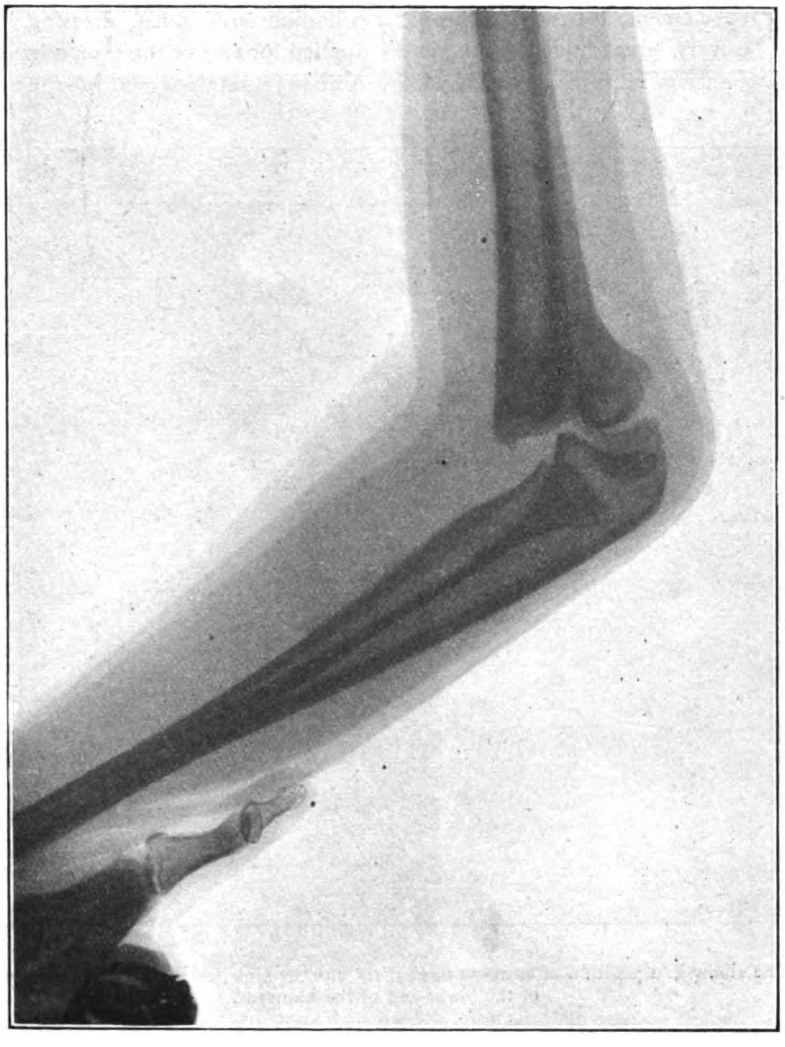

FIG. 15.—The elbow three months after fracture through epiphysial line of lower end of humerus.

is sometimes of triangular or cubical form, and sometimes a partial or imperfect sheath.

Again, in other cases this piece of bone may be loose, having become detached from the epiphysis. The mode of procedure is as follows:—Make an incision along the anterior portion of the deltoid, parallel to and immediately external to the position of the jugular vein, in order to avoid damage to this structure. Then carry your incision backwards along the attachment of the deltoid to the clavicle and to a portion of the acromion. Cut through the deltoid longitudinally, along the direction of the anterior incision, and then transversely about a third of an inch below its bony origin, and turn back the triangular flap of muscle. The seat of fracture is now exposed, and the fragments should be cleared from blood-clot and their margins and form accurately defined. This must be done thoroughly and carefully. By the exercise of considerable traction on the arm, and with the help of

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elevators, you must patiently work the upper end of the shaft into position, taking every precaution not to damage the fragile upper epiphysis or to break away any piece of shaft that may remain attached to it. If there be great swelling of the part, as is so often the case, or if a few days have elapsed since the receipt of the injury, the difficulty of effecting replacement, apparently a simple operation, may be very great indeed. It may sometimes be necessary to remove a portion of a

The wound is then closed accurately, when a perfect result is obtained. In performing any such operations in a part, as the neck or arm, liable to be seen, you should take extreme precautions in bringing the margins of the skin accurately together; and after twenty-four hours or so have elapsed remove the horsehair sutures and apply a collodion and gauze dressing. This should be applied for two or three months afterwards to prevent any stretching and broadening of the cicatrix.

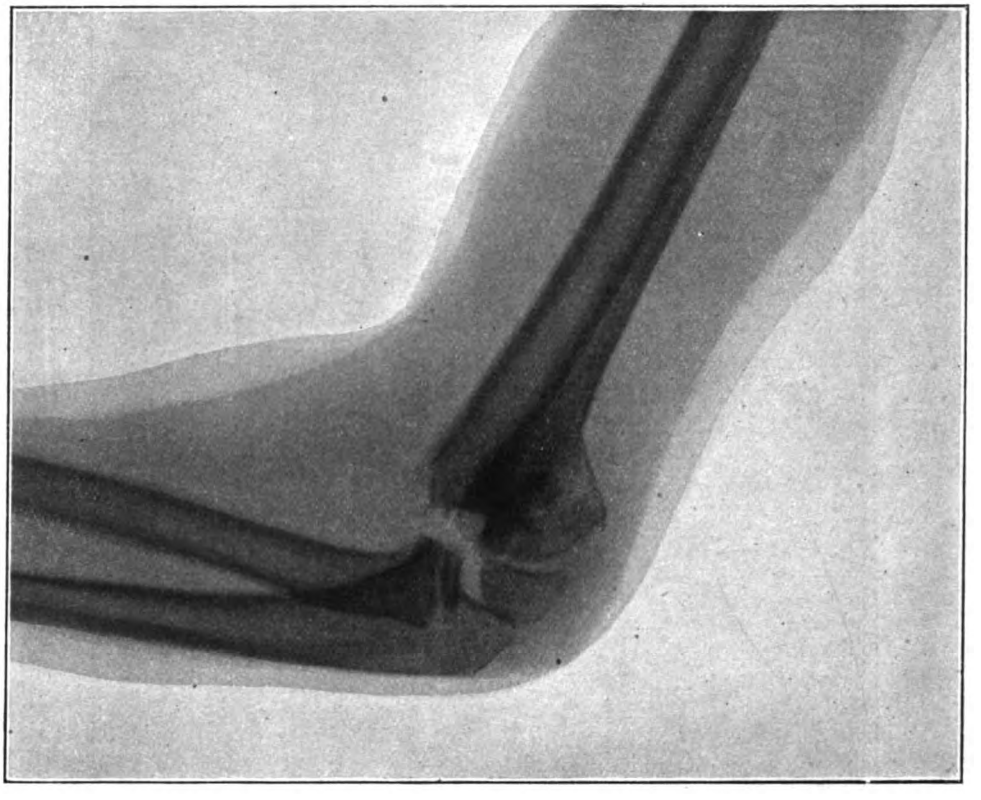

FIG. 16.—The elbow in a position of extreme flexion six months after fracture through the epiphysial line of the lower end of the humerus.

sheath which projects downwards from the epiphysis.

The presence of any piece of shaft in connection with the epiphysis is of great advantage, since it affords a medium by which the shaft may be connected indirectly to the epiphysis by wire. If there be any loose piece or pieces of bone present they should be fitted into place as accurately as possible, and retained there by wire, or a screw, or a tack if necessary.

In fractures of the surgical neck of the humerus the amount of depreciation which follows the ordinary treatment is usually small. Therefore, unless there be any considerable displacement of the fragments on one another, or the perfect performance of the functions of the arm is of great importance to the individual, an operation for fracture in this situation in the adult is but rarely required. The case is, however, quite different when the fracture of the humerus is associated

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with dislocation of the head of the bone. When such a condition exists operative interference is very frequently necessary to effect reduction of the end of the humerus, which is then wired on to the shaft. The procedure is similar to that you adopt in separation of the epiphysis.

Separation of the lower epiphysis of the humerus is unfortunately a very common accident, and one

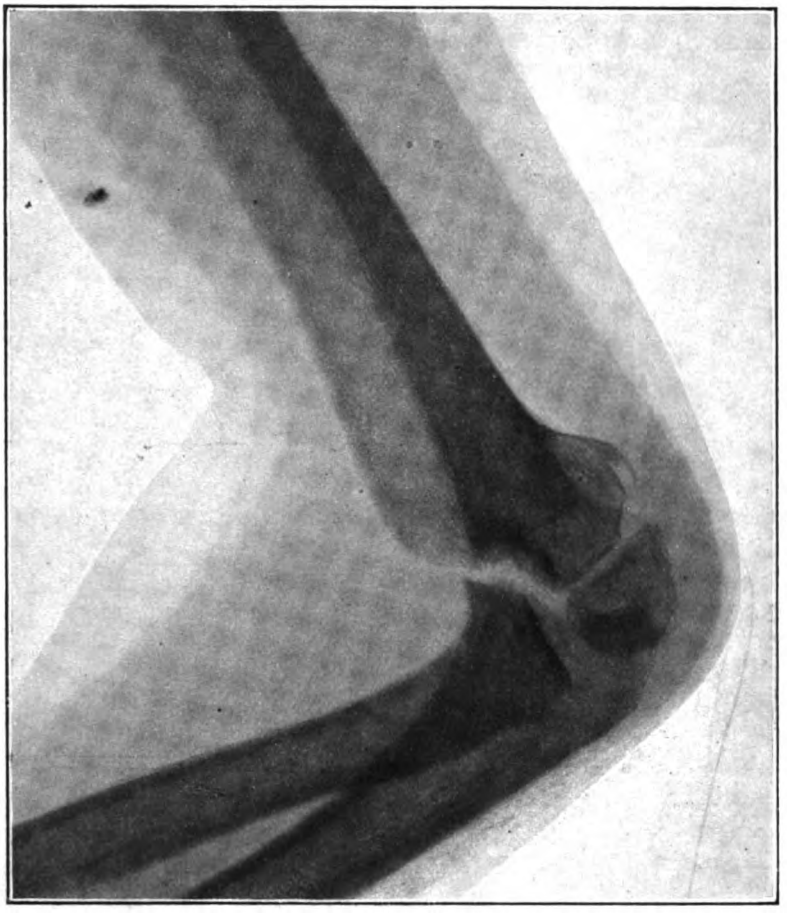

FIG. 17.—The elbow sixteen months after the injury in extreme flexion.

productive of considerable deformity and disability, with a corresponding short life of the elbow-joint, unless the fragments are placed in a fair relationship. Fortunately, as I have shown in a paper published in the 'Practitioner,' September, 1902, entitled "Some Points in the Treatment of Fractures," Nature is able to do a great deal to obviate the result of this injury; since in young life, owing to the formation of callus or new material along the entire length of the shaft, a new shaft is modelled along the line of force while the old one is altered and removed more or less completely.

In other words, at this period of life the scheme of evolution is still maintained, and the new bone represents the crystallisation of the lines of force as compact tissue when it is exerted in one direction, and as cancellous tissue when transmitted in varying directions.

Figs. 15, 16, 17, and 18 illustrate these several changes.

When, in addition to the backward displacement, the epiphysis is displaced considerably outwards, or, as is the case occasionally, inwards; or when the lower end of the shaft has been forced through the muscles ensheathing it, and projects

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beneath the skin, an operation is necessary to enable you to effect accurate coaptation and retain the fragments in apposition.

according to the direction of the displacement; and if there is much swelling and laceration of soft parts, and you are uncertain as to the position of

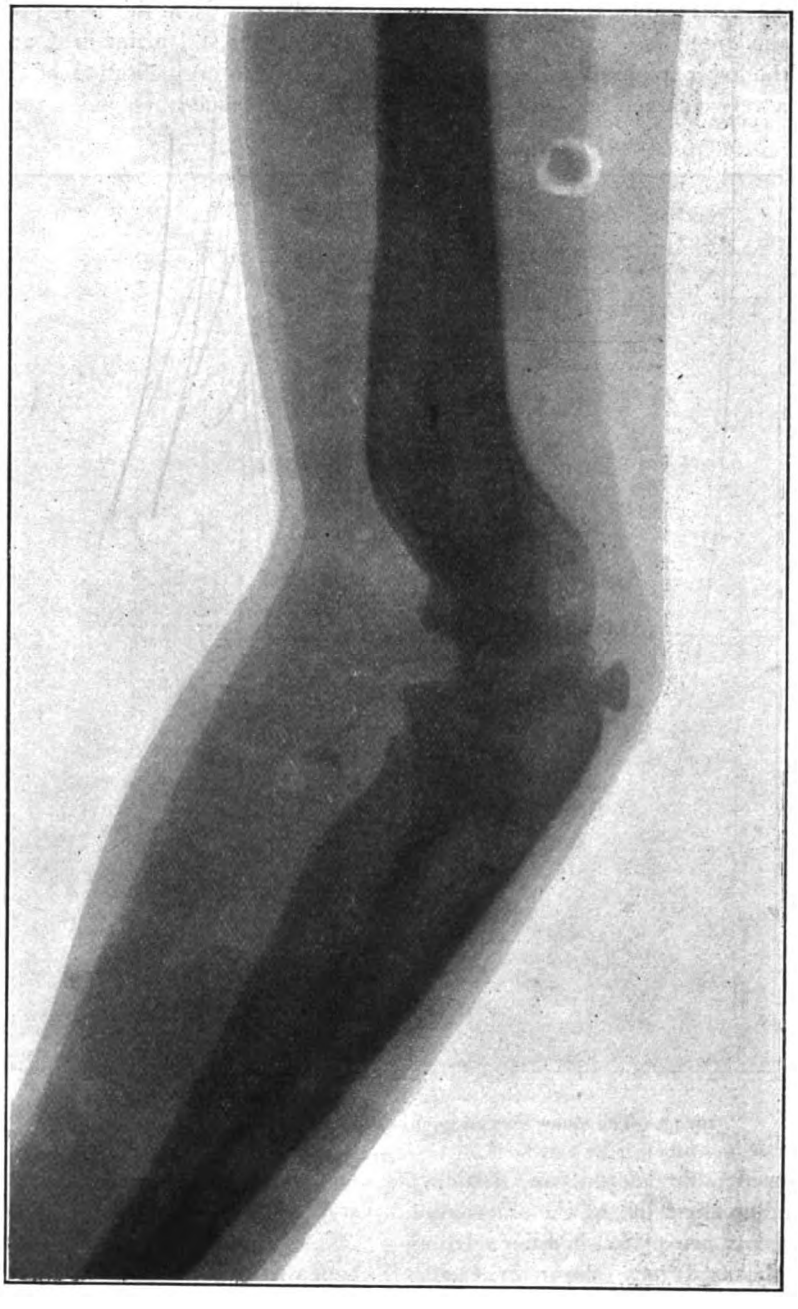

FIG. 18.—The elbow-joint and humerus three years after the injury. The elbow only slightly flexed.

You can expose the seat of fracture by an incision along the inner or outer part of the arm, the several structures, you should define the ulnar or musculo-spiral nerve to ensure its safety.

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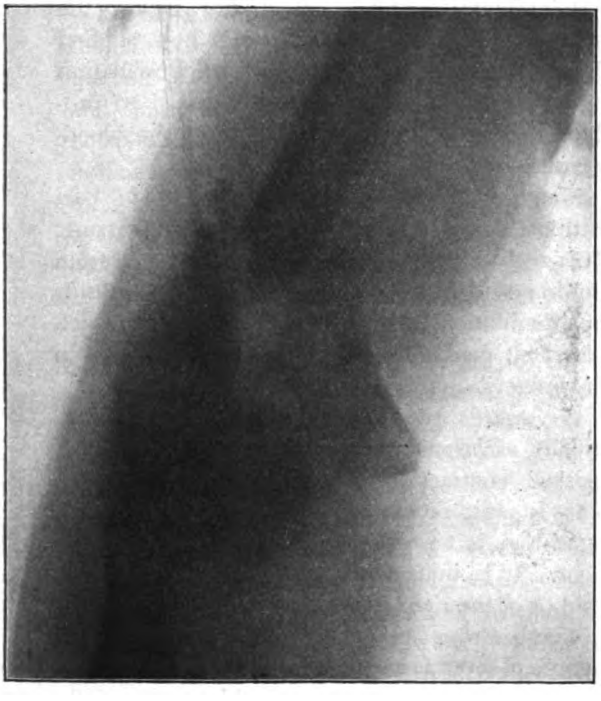
In a fair proportion of cases the epiphysis has attached to it posteriorly a piece of the shaft, which,

FIG. 19.

as in the case of the corresponding injury to the upper epiphysis, serves a useful purpose in holding the wire.

If it is necessary to pass the wire through the epiphysis in the young child, great care must be

FIG. 20.



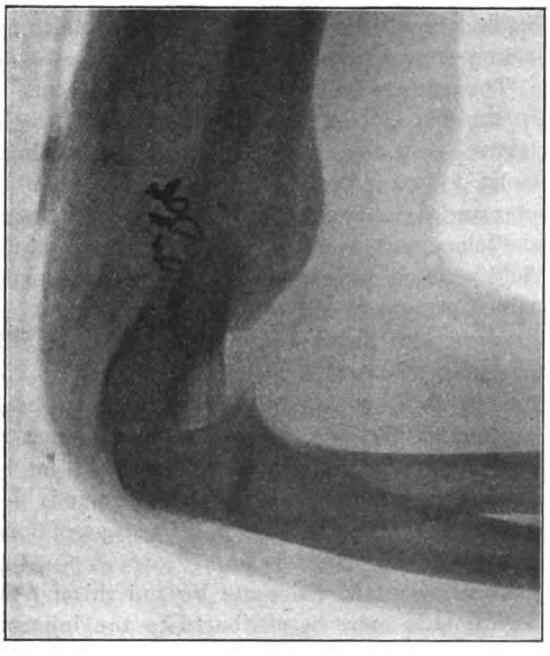
exercised owing to the friable nature of the cartilage and bony centres.

If, after the inward or outward displacement has been overcome, there exists any tendency to backward displacement of the epiphysis, this must be controlled by putting up the elbow in a position of complete flexion, when the tip of the coronoid process impacts on the lower extremity of the shaft and forces it backwards upon the epiphysis. These operations are sometimes very difficult, especially if there is any comminution, or if two or three weeks have elapsed since the receipt of the injury.

Fig. 19 shows the outward displacement which occasionally accompanies the backward displacement of this epiphysis.

Fractures of the shaft of the humerus occasion-

FIG. 21.



ally call for operative treatment, either because of comminution of the fragments, or irreducible forward displacement of the upper fragment in the lower part of the shaft interfering with the proper performance of the functions of the arm, or damage to the musculo-spiral nerve, or the inability to bring the fragments into satisfactory apposition because of the intervention of a mass of muscle through which one of the fragments has been driven, or from any other cause. Fig. 20 illustrates one of the conditions referred to, and Fig. 21 the result of operation. These fractures call for no special treatment beyond free incision and the fitting and securing of the fragments.

(*To be concluded.*)

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WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

GENERAL PATHOLOGY OF FEVER.—II.

THE phenomena of fever which require consideration may be conveniently grouped, as I said last week, under the following headings:—(1) The alterations in the pulse and circulation; (2) nervous phenomena; (3) the disorder of metabolism and respiratory exchange; and (4) the disturbance of temperature.

As regards the pulse and circulation, we must distinguish first between the changes which are attributable to the fever itself and those which are merely secondary to pyrexia.

The increase in the pulse-rate which occurs is, in the first case at any rate, simply due to the increase in temperature of the blood. Even in an isolated frog's heart a definite ratio may be observed, as shown by Lauder Brunton, between the temperature at which the surroundings, and the fluid perfused if perfusion be employed, are maintained, and the rate of the heart-beat—the rate increasing with increase of temperature within the usual limits. And Liebermeister showed that in man there is an increase of about fourteen beats per minute for each degree Centigrade—that is, eight beats for each degree Fahrenheit—of increased temperature. This phenomenon is particularly well illustrated in the early stages of many cases of enteric fever.

Increase in the pulse-rate beyond this physiological ratio must be attributed to the injurious action of the cause of fever, either directly on the cardiac muscle, where it gives rise to the albuminoid degeneration so often seen in cases which have died of febrile illnesses, or indirectly through its action on the cardiac centre in the floor of the fourth ventricle.

Although the rate of the heart-beat increases, the blood-pressure is usually diminished in a case of fever. But this does not occur invariably nor to the same degree in all the fevers; indeed, in certain fevers the blood-pressure may be raised. A sthenic fever is physiologically one in which the blood-pressure keeps high, asthenic fevers those in which it suffers diminution.

The fall of pressure is to be ascribed to a relaxation of the peripheral arterioles. In sthenic fevers with a rapid pulse and very vigorous systole the arterial relaxation may be counterbalanced, or nearly so, by the increased heart's action, the pressure remaining high, or the general arterial relaxation may itself be absent; while, on the other hand, when the arterial relaxation is very marked, or where the cardiac muscle becomes enfeebled by the action of the febrile agent, the fall of pressure is progressively more marked as the systole diminishes in vigour. Between these two extremes, marked fall of pressure from peripheral relaxation, associated with a good flow of blood and a good systole, leads to the phenomenon of dicrotism of the pulse, which is so commonly to be observed in the earlier stages of enteric fever.

The most convenient method for measuring blood-pressure in a fever patient is by means of Mosso's finger plethysmograph, which consists of an upright limb communicating laterally with two chambers, one of which holds the patient's finger, while the other is so arranged that graduated pressure can be applied to the fluid with which the apparatus is filled. The column of fluid in the upright limb oscillates synchronously with the systolic increase and diastolic decrease in the volume of the finger. Pressure is now applied through the second chamber until this oscillation is just abolished. The pressure required to produce this effect gives the measure of the blood-pressure in the finger.

Many of the ill-effects of fever are in part attributable to the lowering of the blood-pressure. This gives rise to a lessened flow of blood from slowing of the general circulation, and hence leads to a diminution of nutrition, the effects of which are often markedly in evidence—for example, in the kidneys.

As regards the vaso-motor changes, there is usually associated with the onset of a fever a marked contraction of the cutaneous arterioles. This is most pronounced where the accession is rapid and is accompanied by a chill or definite rigor. It is, in fact, the cause of the feeling of cold or of the rigor itself.

Traube, who, as we have seen, regarded the pyrexia of fever as entirely due to a diminished loss of heat, laid a great stress upon this spasm of the cutaneous vessels, which he believed persisted steadily until the period of defervescence. This,

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however, is not the case. After the termination of the cold stage there is on the whole a condition of cutaneous vaso-dilatation, though this is variable, and varies in any given area from time to time. Thus at any given moment the vessels in a particular cutaneous area may be found constricted, while those in other parts remain dilated; and in any given part the vessels may be found dilated at one time, but constricted at a subsequent examination. But on the whole the condition throughout is one of cutaneous vaso-dilatation.

Further, the reaction of the vessels of the skin to stimulation or mechanical interference is remarkably slowed. Thus, for example, if an area be blanched by pressure the return of colour takes place much more slowly than in health. This is at any rate partly due to the diminished rate of flow already mentioned.

The defervescence is accompanied by a marked and universal dilatation of the cutaneous arterioles, with restoration of secretory activity in the skin and other evidences of an increased rate of blood-flow, which is probably to be associated with a restoration of control over the peripheral arterioles of the splanchnic area.

We have, then, as the result of the entrance into the circulation of a febrile agent, first a brief contraction of the cutaneous vessels, then dilatation and diminished flow, and finally, in the period of defervescence, a gradual restoration of the circulation to normal. In this connection it is interesting to remember that in the case of inflammation, which is the local reaction to the local introduction of closely similar substances, the local succession of vascular changes runs, as we saw, a very similar course.

The nervous phenomena of fever are to be attributed in part to the actual increase in temperature of the blood and the altered conditions of the circulation, and in part to the direct action of the cause of fever on the nervous tissues themselves. As they concern the higher centres they are chiefly headache, hyperæsthesia of the special senses, apathy, and sleeplessness in milder cases; loss of muscular power, general depression, delirium, and all the symptoms of an intoxication in the severer forms.

As regards so-called "organic centres"—that is to say, those centres whose reflex action is less definitely associated with consciousness than is that of the higher centres, which, for convenience of the heat-regulating reflex, is the most prominent and the most important,—these we shall deal with fully when we come to the discussion of pyrexia. The respiratory centre is also affected by the febrile process, more especially at the onset, when the respiration is increased both in frequency and volume.

This is in part resultant from the muscular activity which asserts itself during the cold stage and leads to an actual increase in the absorption of oxygen; in part it is the effect of increased internal temperature, for the experiments of Fick and Goldstein showed that a heat-dyspnœa is readily produced by heating the blood in the carotid arteries of animals. The cardiac centre may probably be similarly influenced, for there are certain fevers in which the pulse-rate rises rapidly out of all proportion to the rise of temperature, and before any evidence exists of a direct injurious action of the febrile process on the heart muscle—as, for example, in the case of scarlatina. Here the increase is possibly to be ascribed to interference with the normal inhibition exercised by the cardiac centre through the vagus nerve.

The metabolic phenomena of fever are seen in the dystrophia—that is, the diminution of assimilative power—and the disturbance of the excretory processes which are evidenced by the condition of the urine. This is diminished in quantity often to about one third of its normal volume; there is an increase in the nitrogen elimination, not only relatively to the quantity of urine passed, but absolutely, and an alteration in the salts.

There is an accumulation of water in the body during fever. However much is taken by the mouth, less than the normal is invariably discharged. An increased loss of water by the skin occurs, but this is never anything like enough to compensate for its decreased excretion by the kidney. The water thus stored up is discharged later in the epicritical stage following the defervescence.

The diminution in the urinary excretion is due, as was shown by Heidenhain, to the diminished pressure and the resultant diminution of the blood-flow through the kidney. It has also been attributed to the fact that all glandular activity is lessened by fever. But against this explanation is

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the observation already mentioned that the secretion of urea is actually increased.

This increase in urea discharge was first discovered by Traube in 1855. It has been confirmed by Parkes and Ringer and many others. The amount may be as much as from 30 to 50 per cent. greater than in a healthy person on the same diet and under similar conditions.

In health the nitrogenous excretion is almost all derived from the nitrogen of the food ingested in the preceding twenty-four hours, as proved especially by the very exact experiments of Rubner. The excess of nitrogen eliminated in fever can only arise from tissue disintegration.

The daily loss of sodium and potassium salts in health is approximately equal. But in fever the excretion of sodium may be diminished to as little as one sixth the normal amount. This fact was first discovered in pneumonia, and we now know that not only is the normal loss of sodium diminished, but that considerable additional amounts of sodium given by the mouth in the form of common salt may be retained. The excretion of potassium, on the other hand, increases, as also that of the phosphates.

Here, again, we have evidence of the tissue waste which is proceeding, for cells contain an excess of the potassium salts over the sodium compounds, and phosphorus in the form of nucleinic acid and in other combinations. Acetone, another product of disintegration of cells, is also often present in the urine of fever patients, just as in other conditions of rapid cell-degeneration—such, for example, as cancerous cachexia, diabetes, and, as shown by Zuntz, during starvation in the healthy subject. The excretion of kreatin, which, as you know, is probably a product of the muscular metabolism, is also subject to a considerable increase in fever.

We must now pass on from this broad general view to a more detailed examination of the disturbance of metabolism and the pyrexia of the febrile process; and shall consider first the variation from the normal as concerns metabolism.

We have seen that in acute fevers the respiratory movements are increased in frequency and depth, and this implies an increased oxygen tension and a diminution of the carbonic acid tension in the blood, supposing the rate and volume of the circulation to remain the same. But with the fall of the blood-pressure in fever the blood-flow becomes slowed, a condition naturally tending to increase its venosity by increasing the time of the circulation, and therefore the intervals during which any given volume of the blood is absent from the lung capillaries. Some part of the increase in the respiratory movements, therefore, may be dyspnœic, and due to an excess of carbon dioxide in the circulating blood. It certainly is so in fevers which deteriorate the functional activity of the lung tissue, though even here the greater part of the increase is to be attributed to the other causes which we have already mentioned—namely, the action of the superheated blood and of the fever-producing agent on the nervous centre. That this is so is evidenced by the rapid fall in respiration rate with the crisis of pneumonia, although, of course, no sudden change in the condition of the lung itself occurs.

But whether the blood be slightly more arterial than normal, as in the early stage of increased respiration and before the fall of blood-pressure and the slowing of the circulation has begun, or slightly less arterial than normal, cannot materially affect the respiratory exchange, since, as you know, the respiration of the tissues is intrinsic, and is not immediately dependent on the blood condition. It is the rate of tissue metabolism itself, and the requirements of the various tissues which determine how much oxygen is taken up from the blood, and how much carbonic acid gas is formed. Accordingly it is the actual respiration of the tissues which requires to be determined in investigating fever.

Now Liebermeister found in his experiments that in the initial stages of fever the respiratory exchange may be as much as twice or thrice as great as normal. But Senator, in investigating the same subject, came to the conclusion that the increase in the discharge of water and carbon dioxide by respiration was never greater than could be accounted for by the increased activity of the respiratory movements. Leyden, however, repeating the experiments some years later, found in all cases accompanying the rise of temperature an increased discharge of carbon dioxide of such amount and duration as could not possibly be explained by the increased respiratory movements alone. And his results were supported by the experiments of Finkler working under the great physiologist Pflüger.

A brief increase in the elimination of carbonic

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acid could be accounted for by increased activity of respiration leading to a more rapid discharge of the carbonic acid already accumulated in the blood and tissues; but a considerable and continued increase must imply increased production of the gas in question by increased metabolism.

After the invention by Professor Zuntz of a much more accurate and convenient apparatus for determining respiratory exchange in man than had previously been available for the purpose, and the exact investigation made by himself and Lehmann on the exchange of gases in the two fasting men Cetti and Breithaupt, the subject of the exchange in fever was again taken up by Kraus and also by Loewy. The general results which they obtained may be summarised as follows:—(1) There is usually increase of the intake of oxygen and of the elimination of carbon dioxide with or without increase of the respiratory movements. But this only occurs while the temperature is rising during the accession, and is associated with the shivering and increase of muscular tone which present themselves during this stage. (2) The respiratory quotient—that is, the ratio $\frac{CO_2 \text{ discharged}}{O_2 \text{ taken in}}$—is in many cases unaltered, but it may be diminished. Such diminution, however, is not a pathological event, since it is present equally in healthy persons kept on fever diet and under the same general conditions.

On the whole, therefore, the respiratory exchange is unaltered in fever. Such variations as may be noted occur only in the early stage, and are secondary to an increased metabolism of the muscles, and do not constitute an essential or invariable character of the febrile process. Accordingly there is no increased consumption of fat in fever, since this we know would show itself in an alteration of the respiratory quotient.

Following the discovery of Traube of the increased elimination of urea in fever came a series of investigations on the discharge of nitrogen both in man and fevered animals by Ringer, Naunyn, Senator, and many others. Huppert had early shown that on the whole the nitrogenous excretion varies with the temperature, and Ringer found that the increased discharge commences even *before* the rise of temperature at the onset of the febrile process.

The discharge of nitrogen in fever is equal to or often even greater than that which occurs in health, and is frequently more than double what it would be in a healthy person on the same diet and under the same conditions as the fever patient. It is, therefore, clear that tissue proteid is being broken down in fever; and this is evidenced again, as we have seen, by the increased potassium, kreatin, and phosphates of the urine, and in the appearance of acetonuria. What is the cause of this increase of the metabolism of the tissue proteids?

There are several possibilities before us. It might be due (*a*) to a direct action of the cause of fever on the tissue-cells producing an increased lability of tissue; or (*b*) to the action of the same agent on a trophic nervous centre, either in the direction of stimulation or of a paralysis of normal trophic inhibition; or, again, (*c*) to the action of the increased temperature in accelerating metabolic change; or, lastly, (*d*) to the dystrophia associated with the fever, which, by preventing normal assimilation, forces the animal body to carry on organic processes at the expense of its own tissue substances.

Now to examine the causation of a result in which several variable factors may take part, the proper course is to determine the result of varying each factor separately. We thus determine what part if any of the general result depends on any one particular factor.

Proceeding in this way, we must examine first the condition of inanition which most closely resembles in its physiology the dystrophia of fever. Here, as regards the human subject, very exact results have been presented by the observations of Zuntz and Lehmann on the fasting men already mentioned. In animals the very accurate experiments of May on rabbits, published in 1894, furnish the most reliable data available.

Taking an animal in a condition of inanition—that is to say, in fasting,—it is found that after the first few days, in rabbits by the third day, the daily loss of nitrogen and of carbon becomes very nearly constant. This point corresponds to the period when the food last taken and the reserve of carbohydrate in the form of glycogen has become used up. The animal has now to live on its own body tissues. Hence from its daily loss of nitrogen we can determine the amount of tissue-proteid waste, since fifteen parts of nitrogen correspond, as we know, to about a hundred parts of proteid matter. Having thus calculated the amount of proteid

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broken down, we determine next the amount of carbon it contained, and, subtracting this from the total carbon eliminated, obtain the quantity of carbon which has been derived from fat, and thus the amount of fat used by the animal.

In this way May found that in eight rabbits which he employed for the experiment the daily elimination of nitrogen was about 0·055 per cent. of the body-weight. When, however, a condition of fever was induced in the animals in addition to the inanition, the loss of nitrogen increased, and amounted, for example, to 0·066 per cent. of the total body-weight. But the consumption of fat was found to be unchanged—a result confirming the conclusion which we have already reached from the consideration of the respiratory exchange. These results are well illustrated by a table from one of the analyses prepared by May, which is as follows:

Condition of rabbit.	Daily loss of nitrogen.	Proteid equivalent.	Carbon in the proteid.	Total loss of carbon.	Carbon lost in fat used.	Fat used.
Inanition	2·1 grms.	14·0 grms.	7·4 grms.	15·0 grms.	17·6 grms.	10·0 grms.
Inanition *plus* fever . . .	2·7 „	18·0 „	9·7 „	17·3 „	7·6 „	10·0 „

Proteid waste increased nearly 30 *per cent. by fever; fat used not increased in amount.*—It follows that the nearest approach to the metabolic conditions which obtain in fever is to be found in the condition seen in inanition, the only difference being that in the former an increased amount of tissue proteid is used. Dystrophia, therefore, as we must conclude, is the prime factor in the metabolic disturbances of fever. Only the excess of proteid waste in fever over the tissue-proteid waste of inanition can be ascribed to causes other than the nutritive disturbance. And this excess may amount, as we have seen, to something like a 30 per cent. increase on the nitrogenous metabolism of inanition.

What is the cause of this increase in fever? It might be accounted for in part by the pyrexia. All chemical processes go on more actively at a high temperature than at a lower, and we should therefore expect more active tissue change when the body temperature is raised than in a state of inanition, which is usually associated with subnormal temperatures. Moreover exact experiments on hibernating animals, and especially those carried out by Dr. Pembrey, have proved that diminution of metabolism occurs in hibernation in consequence of the lowering of the animal's temperature. High temperature alone, however, cannot account for the excess of the nitrogenous metabolism in fever over that of inanition, since, as we shall see in the discussion of pyrexia, there is probably a nervous regulation tending to diminish metabolism and thus lessen the heat-production of a normal animal should the conditions tend to cause a rise of body temperature which is insufficiently controlled by thermolysis.

We are therefore driven to ascribe the excess of proteid waste in fevered animals to the direct influence of the cause of fever. And this position is confirmed by the observation of Ringer that the increased excretion of urea begins *before* the temperature rises at the onset of the febrile process; that it is greatest at an early period in the fastigium—that is to say, when the reserve of carbohydrate in the body is used up, and the activity of the febrile agent has presumably reached its height,—but that it declines towards the end of this stage and through the defervescence when the action of the causal agent is again subsiding. The proteid disintegration is, however, apparently of the same nature as that of inanition, and its end products, including acetone, are identical with those observed in an afebrile wasting illness, and in the experiments on fasting men already mentioned. Moreover May has shown that as much as 25 per cent. of this nitrogenous elimination in fever may be saved by giving carbohydrate in the immediately assimilable form of glucose—a fact which has not as yet received the clinical recognition it deserves.

Now just as there was found in inanition the most useful guide to the condition of nutrition in fever, and one which made it possible to examine the condition of dystrophia with or without an associated febrile process, so the experiments of Kraus and Loewy with tuberculin both in the

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human subject and in animals exhibited the metabolic changes of febrile pyrexia apart from those associated with dystrophia. These observations made it clear that increased proteid waste accompanies the action of a febrile agent. The injection of tuberculin in tuberculous individuals gives rise to fever without important alteration in the digestive and assimilative functions. It none the less occasions an increased excretion of urea. And hence it follows that the excess of proteid waste in fever over the proteid-tissue waste of inanition is due directly to the action of the cause of fever.

Whether the febrile agent acts immediately upon the tissues or only mediately through the nervous system cannot perhaps be definitely answered in the present state of knowledge. But while, as we shall see, there is, as yet at any rate, no *proof* of the existence of a central nervous mechanism for heat-production, nor of the existence of a system of trophic nerves by which that mechanism could control the tissues, there is, on the other hand, abundant proof in the phenomena observed in local inflammations that morbid substances which gain a local entrance can produce tissue disintegration by direct action on the cells concerned. Such substances, moreover, are akin to those which bring about the febrile process.

During the defervescence the nitrogenous waste diminishes, but following it there frequently occurs an epicritical increase, as it is called, in the elimination of urea in the urine. This is to be ascribed to the increased assimilation which occurs, and is the ordinary physiological result of increased diet, for, as you know from the experiments of Voit and others, if an animal be in a condition of nitrogenous equilibrium or below this level, an increase in its consumption of proteid food invariably leads in the first case to an increased destruction of proteid in the body, and an increased excretion of urea, greater than corresponds to the increase of proteid assimilated.

We have seen that an animal in fever has an increased excretion of urea over the excretion by a normal animal on the same diet; this means increased production of heat from proteid metabolism, and this implies greater facilities for increase of body temperature or pyrexia. All this, however, is insufficient to explain the rise of temperature, for in the normal animal an increased supply of heat is counterbalanced by an increased loss under the action of a nervous regulation. This regulation we must now consider.

The process of heat-production is spoken of as thermogenesis, that of heat-discharge as thermolysis, while the regulation by the central nervous system of these two opposing factors to maintain a constant and a stable body temperature is called thermotaxis.

The stability of the body temperature in health is quite as marked and definite as its constancy. And the importance of this character was especially emphasised by MacAlister in his Goulstonian Lectures upon fever. Man is not only homoiothermic, but also thermostatic. Not only does the temperature in health tend to maintain a constant level or normal, but it also tends to rapidly regain this normal level if it be temporarily diverted either up or down by outside agencies, and it is only with considerable difficulty that it is thus diverted. The temperature of fever, on the other hand, is not only not constant, but it is also remarkably unstable. Very slight changes in the external conditions produce considerable variations, and the return from such a variation is not only slow, but often also very incomplete.

It is, of course, familiar to you how easily a fall of temperature can be produced by tepid sponging in continued fevers, and how comparatively slowly it regains its former level subsequently; and, on the other hand, what very slight disturbances may cause a rise in temperature of one or two degrees. Accordingly the view of Liebermeister that in fever the temperature is merely adjusted to a new "norma" is untenable, for adjustment or regulation means stability as well as uniformity, while stability is conspicuously absent from the temperature of fever even in those cases where the pyrexia of the fastigium usually maintains a fairly constant level.

Such uniformity of temperature as occurs in fever is therefore not dependent on an adjustment of the regulative mechanism to a new level, comparable to the readjustment of a thermostat by a slight alteration of its regulator—a mystical change, as Cohnheim put it in his criticism, from the human regulation to the regulative level of a bird. Liebermeister's view would make the process comparable to the tuning of an instrument to a higher key, where all the relations of the different parts remain unchanged and the mechanism quite

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unaltered in its accuracy, only the general level of its action being raised; whereas in fever the elevation of the general level is only secondary, and is the outcome of a condition of disorder in the mutual relationship of the different factors which take part in the production of the body heat. Whatever uniformity may be observed in the temperature of the fastigium of continued fevers is rather to be correlated with the fact observed by Wood, of Philadelphia, that the production of heat during this period of the febrile process is fairly constant, and is not a markedly fluctuating quantity.

The regulation of the production, distribution, and discharge of body heat so as to maintain a uniform body temperature is characteristic of the higher Vertebrata, which are accordingly spoken of after Bergmann as homoiothermic animals, in contradistinction to the poikilothermic animals, whose temperature is variable and varies with the temperature of their surroundings. This does not mean that the so-called cold-blooded animals have a temperature identical with that of their surroundings. Their own production of heat in the course of their metabolism prevents exact coincidence with the external temperature. What is implied is that in these animals the regulation is rudimentary, or at any rate imperfect, so that increase of the external temperature induces increase of their metabolism, and a decrease of temperature a diminution in its activity, as shown by Pembrey and by Vernon. The relations are well seen in the following table, which I have taken from Landois and Stirling:

Frog in water.		Frog in air.	
Temperature of water.	Temperature of frog's stomach.	Temperature of air.	Temperature of frog's stomach.
41·0° C.	38·0° C.	40·4° C.	31·7° C.
30·0° C.	29·6° C.	27·4° C.	19·7° C.
20·6° C.	20·7° C.	16·4° C.	14·6° C.
5·9° C.	8·0° C.	6·2° C.	7·6° C.
2·8° C.	5·3° C.	5·9° C.	8·6° C.

Homoiothermic animals, on the other hand, are able to diminish their production when the external temperature rises to such an extent as to seriously check the surface loss of heat, and to increase it when external cold leads to excessive loss of heat sufficiently to maintain an almost constant level under all ordinary variations of their environment. And moderate changes of external temperature do not affect their rate of heat-production at all, being entirely dealt with by the regulated loss of heat.

The hibernating animals form an intermediate class, maintaining in their waking periods a uniform and stable temperature, but during the winter sleep reacting like cold-blooded animals to variations in external temperature.

Temperature regulation to a constant level is thus peculiar to the higher animals,—that is to say, it is of late development in evolution. It is also of late development ontologically in the individual. Thus, as was shown by Pembrey and Gordon, the embryo chick reacts as a poikilothermic animal to variations of external temperature, the chicken only gradually acquiring homoiothermic characters within the last few days before it leaves the shell. And probably the human infant is at birth and for some period afterwards quite incompletely capable of homoiothermic regulation of its body temperature.

This regulation is controlled by the nervous system; and we may here recall in this connection the established observation of the late development of the functional activity of the higher nervous centres, and the late acquirement of their medulla by the cortical fibres and the sensory tracts, which probably contain the afferent conducting paths for heat and cold. For, as we shall see, the regulation concerns the higher centres, and is to some extent under the control of individual volition,—that is to say, the reflex is in part one of the so-called psychical reflexes.

THE value of quinine salicylate in rheumatic affections, and its occasional usefulness in influenza, diarrhœa, and neuralgia, are well recognised. For some years a "Tabloid" product, containing three grains of the salt, has been issued by Messrs. Burroughs Wellcome and Co., but as it is sometimes desirable to prescribe smaller doses, more frequently repeated, "Tabloid" Quinine Salicylate, gr. 1, is now prepared, and is issued in bottles of twenty-four and a hundred.

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A CLINICAL LECTURE

ON

TUBERCULOUS PERITONITIS.

Delivered at St. Bartholomew's Hospital, May 15th, 1903.

By SIR DYCE DUCKWORTH, M.D., LL.D.,

Physician to the Hospital.

GENTLEMEN,—I want to-day to call your attention to the subject of tuberculous peritonitis. I have had one patient brought down whom it is very important you should see. He is a somewhat sickly-looking lad, rather pallid. He was admitted on March 25th, and the complaint was that he had been suffering from pain in the abdomen for some time since Christmas. His history went to show that, except for a slight cough, which he had for eighteen months, he was in enjoyment of average health. Since last Christmas he has had abdominal pain, chiefly in the lateral region. The pain was continuous day and night, and was increased after food. The bowels acted daily, but there was no diarrhœa and no constipation. The appetite has been good, and there is a history of night-sweats. Since Christmas he had noticed that the abdomen had been increasing in size, and that he had been getting thinner. Six years ago he had an attack of pneumonia, and had had bronchitis twice previously to that illness. With regard to his family history, his father is a healthy man, but two of his father's sisters and the paternal grandfather were consumptive. The members of his mother's family were healthy. He has got another brother who is also healthy. On admission he had a pulse of 116, regular, of moderate volume and low tension. The respirations were twenty per minute. On examination of his chest on admission there was seen to be diminished expansion in the upper part of the right front, in which situation a few crepitations were heard, more especially at the right apex. The heart-sounds were natural. The abdomen was noticed to be very full. There were

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no enlarged veins upon it. It was resonant all over except in the flanks, and the resonance was altered when the patient was turned on to his side. There was no œdema or redness of the abdominal wall, and no particular tenderness on palpation. We will look at his abdomen now. You will notice that it appears full, and one or two veins can be seen crossing over it which are perhaps a little fuller than they should be. A very distinct wave of fluid can be obtained. On percussion over the front part a resonant note is elicited, but down in the flank the percussion-note is dull. With this distension there is some ascites. On examining generally all over the abdomen one finds it is very hard and resistant; there is a tough hardness all over. The muscles are tense, and appear to be guarding the abdomen. There are no hard masses to be felt, and no indurated thickened regions. Of course it is possible that there may be masses within the abdomen which the rigidity of the muscles prevents our detecting. It is quite certain, however, that there is fluid. We will now look at his legs. His general nutrition is very poor. Slight dimpling is just possible over the tibiæ. These are all the physical signs which we can find now, but when he came in there was evident consolidation of the lungs, more especially at the right apex. Since his stay in the hospital, however, these signs have subsided, and now the lungs seem to be in a fairly satisfactory state. The diagnosis in a case of this kind is not difficult, and not far to seek. In arriving at a decision the history goes for a good deal. You will have noticed the gradual and insidious onset of his illness, beginning by poor health and pain, together with enlargement of the abdomen. Further examination shows indications of disease of the lung, more especially the right one. Putting these factors together, the case becomes about as clear as you could desire—namely, one of tuberculous invasion of the peritoneal cavity, which is recognised as tuberculous peritonitis. It is a fairly common complaint, occurring chiefly in young people, but it may be met with at any age. Certainly it is more common in the male sex than in the female. It is interesting because this condition of the abdomen constitutes the most favourable form of tuberculosis which we encounter. You have only to contrast it with tuberculosis of the lungs, or with that gravest of all forms of tuberculosis, which involves the membranes of the brain—namely, tuberculous meningitis, one of the most deadly diseases which afflict humanity,—to see how different is the outlook. Having made the diagnosis, we have now to consider, in a lecture like this, how it comes about. Where does it come from? We have found that it is accompanied by pain in the first instance, by poor health, by wasting, and sometimes by tenderness (though there does not appear to be any tenderness of the abdomen in the present case), with indications of fluid, and in many cases, though not in this, the occurrence of large lumps in the abdomen. The peritoneal and visceral membranes become studded with tubercles. These may appear as fine grains like sand, called miliary tubercles, or more commonly, when the disease is not acute, be characterised by lumps of tuberculous matter which caseate, and are very apt to involve the great omentum. And as this deposit and exudation go on, due to the tuberculous invasion, the omentum becomes puckered up into lumps and masses, sometimes feeling like a coil of rope across the abdomen. Occasionally, that is noticed to such an extent as to lead to the belief that there is enlargement of the liver. But that idea can generally be corrected by examination of the whole abdomen, inasmuch as you would expect to get resonance above a hardened coil of omentum and between it and the dulness proper to the liver. So you may expect to meet with cases in which there is a good deal of effusion, and with others in which there is less or no effusion, characterised by lumps in the abdomen and actual retraction of the abdominal wall. Thus the disease assumes different phases according to the amount of effusion, the presence or absence of it, or according to the occurrence of these caseous lumps within the abdomen. As you probably know, just now a dispute is going on amongst bacteriologists as to the origin of abdominal tuberculosis, especially in children and young people. The question is whether tuberculosis is capable of passing from the lower animals to the human subject,—that is to say, passing by means of meat or milk, for the question hangs chiefly upon these two substances. The adherents of one school, led by Professor Koch, say that the risk is *nil*, and that bovine or animal tuberculosis is not communicable to the human subject.

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Others, like Professor Hamilton, who has re-investigated the subject since Professor Koch's statements in London here last year, declare that tuberculous meat and milk may communicate tuberculosis to the human subject. In the meantime, the majority of authorities hold the opinion that the disease is transmissible from animals to man, and that therefore it is necessary to take the prescribed precautions in respect to the thorough cooking of meat—or not consuming it at all if it is likely to be tuberculous—and boiling the milk, or at all events raising it to such a point that any bacilli in it are destroyed. I have no doubt that the question will be settled for certain before long. In the meantime, in whatever way the bacilli get into the body in such cases, they find a suitable and appropriate soil. It is not possible for a person to become tuberculous unless he is predisposed to it,—that is to say, unless his bodily conformation and the quality and condition of his tissues are suitable for the bacilli to grow in. The persons who present this suitable soil are those of strumous constitution. But you will remember it is possible to be strumous without being tuberculous. That is a doctrine which I hold, and teach, because I maintain that it is possible to be strumous throughout the whole of life without ever becoming tuberculous; albeit a strumous person is much more liable than any other person not strumous to become the subject of tuberculosis. Whether the bacilli get in by way of the tonsils and thereby become deposited in the glands, or whether the bacilli are conveyed directly into the abdomen by means of food, is uncertain; but at all events they lodge in the abdomen and peritoneal cavity of persons predisposed to harbour them. And so we find either that the mesenteric glands themselves become infected, as a sort of storehouse of tubercle, and proceed to infect the peritoneum directly; or it may be that a focus is set up in the mucous membrane of the alimentary canal, and the bacilli pass through the bowel. Dr. Sidney Martin has shown that the bacilli can pass directly through a healthy intestine into the peritoneum without any breach of surface existing. He has proved that to be the case in rabbits at all events In other cases where tuberculosis of the lungs exists the bacilli may readily be carried by the lymphatics or blood-vessels into other parts of the body, and so reach the abdomen; or, as often happens in females who become the subjects of tuberculous peritonitis, the disease arises first in the Fallopian tubes or uterus. In the male it may start from tuberculous testes. Or, again, the process may start in a tuberculous hip-joint. By a consideration of the case and inquiry into the antecedents and the family history, you will hardly ever be in doubt in any case of obvious chronic peritonitis occurring in such a patient as we have here that it is one of tuberculous origin. Some cases are exceedingly acute, and may quickly pass beyond the reach of treatment, the entire peritoneal cavity becoming studded with miliary tubercles. But many cases of tuberculous peritonitis occur in which the lungs are hardly involved at all. Or, again, you may find old cicatrices of past tuberculous disease when making a *post-mortem* examination years after the tuberculous affection has become arrested. It is more common to have the abdomen involved without the lungs being infected, than to find both lungs and peritoneum involved. Of the class of case now before us one happily is able to think very favourably. On the whole they are not unsatisfactory cases; one's concern is less for those who present tubercle in the abdomen than in any other parts, and the reason is that these cases when carefully treated and watched are found to get quite well. What happens is that under the influence of appropriate treatment, good food, and good healthy surroundings, the effusion, if there be any, dries up, and any masses that there are become less and less, and actually obsolescent; and many years afterwards, if a *post-mortem* examination is made of such cases, there are found to be hard, cheesy masses which indicate that there has been tuberculosis there at one time. I have not mentioned that a considerable degree of fever is apt to occur in these cases. The chart of this boy when he came in showed a temperature which used to rise every night over 103°, and that went on for some weeks. Under the influence of treatment he improved so much that his temperature fell and became moderate, and it remains so; he has lost his high temperature.

You would now like to know what we did for this boy. First we gave him a slop diet, with strong essence of beef; then we gave emulsion of cod-liver oil three times a day, and a preparation of iron and manganese, particularly phosphate of iron. The local treatment, on which great stress must be laid for this condition, was to cover the abdomen with

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a preparation of oleate of mercury (10 per cent.) and extract of belladonna, equal parts. This preparation was put on lint or flannel, and bandaged across the abdomen. After a while he was able to take an improved diet, including some wine. Then we changed the mercurial preparation for another—namely, the common blue mercurial ointment. Gradually we found that the effusion diminished; the girth of the abdomen is diminishing, and we regard him as being well on the way to recovery. Had the effusion materially increased instead of diminishing, I should have been prepared to do something more; I should have asked Mr. Marsh to consider the advisability of opening the abdomen—that is to say, to perform laparotomy,—but only with the object of liberating the fluid and closing up the abdomen again. That is a method of operation which is of decided benefit, and it is one which may be practised whenever necessary. It may seem a strange measure to adopt, but it arose in this way:—Forty years ago the late Sir Spencer Wells opened the abdomen of a young woman who was supposed to have an ovarian tumour. She had a large lump in the lower part of the abdomen, and all her symptoms pointed to ovarian tumour, which he proposed to remove. At that time ovariotomy was coming into vogue very much; and he was accumulating his statistics, which embraced over a thousand cases, and his experience established the certainty of the operation. Hitherto it had been one of the deadliest operations in surgery. Having opened the abdomen of this particular woman, he found no ovarian tumour, but the whole peritoneum and the surfaces of the bowel were studded with numerous tubercles. He therefore simply let out the fluid, sponged over the intestines, and sewed up the wound, expecting that in thirty-six hours or so the poor girl would die. But on the contrary she steadily improved, and eventually got perfectly well; and after six years she was a healthy, stout, robust woman, and was married. It was very difficult to offer any explanation, but it made people think. It seemed that the evacuation of this fluid started a healthy process, which resulted in the clearing up of the tuberculous condition, and upon the experience of this case people were led to tap the abdomen in this condition, and the results have practically always been advantageous. Of course the laparotomy is done on the ordinary principles of clean, aseptic surgery; and, as I have said, if the course of this case had not been favourable I should have been quite prepared to have had that procedure adopted. In other cases of tuberculous peritonitis the fluid takes on other qualities; it does not remain serous or slightly blood-stained, but passes into pus, so that you then have a peritonitis depending upon broken-down tubercles. There is continued fever in these cases, and generally speaking there is some determination towards the umbilicus. It is very apt in these cases where it takes this suppurative turn to produce an inflammatory process about the umbilicus, a ring of redness and hardness of the abdominal wall at that point, sometimes the thickening passing up from the pubes along the urachus. Then there is bursting of the integument near the umbilicus, and the issue from it of purulent fluid. In that way the abdomen empties itself spontaneously, and very favourable results have followed this opening of the abdomen by Nature herself, whether the fluid be simple or whether it be purulent. All that is required in that case is to keep the parts very clean so as to prevent septic contamination. Of course the general health has to be attended to, and the treatment is the same as for tuberculosis anywhere—namely, cod-liver oil and iron. When it can be arranged, this treatment should be carried out in strong sea air. This boy we shall try to send to Margate or some good seaside resort. I believe Margate to be the best for all strumous people, and certainly such people get better there quicker than at any other seaside resort that I know of. As I have said, there was a suspicion of the apex of one lung being involved in this boy, but that appears to have got better, and if no further deposits occur we may expect complete recovery.

In the same ward with this boy is a man æt. 24, an agricultural labourer, who came in on April 24th. Having regard to all his symptoms and his history, it seems more than probable that this also is a case of tuberculous peritonitis. When admitted he complained of abdominal pain. He was in his usual health up to Christmas-time, when he began, without any apparent cause, to have pain in his abdomen. This was of a dull aching character, and was unaffected by taking food or liquids. There was constipation, but no vomiting. He is said to have been consumptive when he was

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fourteen years of age, which is ten years ago. He appeared to be fairly healthy looking, but spare, when he walked into the ward. His pulse was of fair tension, chest well formed, nothing wrong with his heart, but a little dulness at the apex of the right lung posteriorly, although the sounds were clear on auscultation. The abdomen was found to have its muscles well developed, and they were rigid under examination. No lumps were felt in the abdomen nor sign of fluid, and there were no enlarged viscera. There was nothing remarkable about his legs. The condition of his abdomen has been found to vary. Since he came in he has had marked fulness in the epigastric region, but there was nothing definite to be felt except that the muscles were contracted, indicative of some tenderness underneath. But we know that his temperature is raised every night to 101°, and his pulse-rate is over the average—namely, 96; the respirations also are a little quicker than those of a healthy man. There is nothing more to be observed in the case. But there is enough in all that I have told you—his personal history, his family history, and his symptoms—to justify the opinion that this also is a case, though it may be a very mild one, of tuberculous peritonitis. If it is not that, it is very difficult to say what it is. Some of these cases are so obscure in the first instance that it is impossible to be certain or to make a diagnosis. You will remember that a diagnosis is always improved by time. Time clears up difficulties, and if you can only wait long enough you can generally overcome them. Medical men are sometimes hardly dealt with in chronic cases because of this fact. The first doctor called in perhaps finds a difficulty in arriving at a diagnosis. As times goes on the recognition of the disease becomes a little easier, though it may still remain a difficult problem; but the patient does not understand the doctor's difficulty. He thinks the doctor exists for the purpose of finding out at once, and determining exactly what is the matter with each individual; and if he cannot do it someone else must be called in. Accordingly another opinion is sought for. The case may still be difficult; the friends are not satisfied, and in course of time a third opinion is asked from another medical man, and this third opinion is generally found to be quite satisfactory. The cleverest physician always arrives last, and he makes the diagnosis. The fact is, however, that the nature of the disease has been becoming more and more obvious every week, when at first it was not at all clear.

One other remark I have to make, and that is this:—As I have often told you at the bedside, if you have an obscure case in early life, or up to the age of thirty, a clinical puzzle which you cannot easily solve, in which you say the disorder does not seem to conform to anything you know of, and yet the patient is very ill; it cannot be this, it is not that, or the other,—it will nearly always be found to be some form of tuberculosis. It is an important thing to remember in practice that many of these puzzles at that age-period can be put into the category of tuberculous disease.

These two cases are being treated alike, by means of tonics and cod-liver oil, and we shall try to get them both to the seaside, which is the great healer for all tuberculous ailments. Remember that tuberculous peritonitis is the form of tuberculosis which we are most hopeful about. We expect sufferers from it to get well, provided there are no complications in other parts, or any ulceration of the bowel, or progressive tuberculosis in the lung. When tuberculosis is confined to the abdominal cavity we may hope that the patients will recover, and probably live to a good old age.

The Treatment of Typhoid Fever by the Flowers of Sulphur.—Caramano states in the 'Journal des Praticiens' of November 29th, 1902, that the flowers of sulphur have been employed with advantage in the treatment of typhoid fever, the dose amounting to about 15 grains every two hours. These doses are said to be taken without any inconvenience, and are particularly valuable in those cases which suffer from constipation. The advantages claimed for this method of treatment are that while it diminishes the temperature it also produces a general improvement in the condition of the patient. It also exercises a favourable influence upon the intestinal mucous membrane.—*Therapeutic Gazette*, June, 1903.

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THE JUDICIOUS USE OF TONICS.

By CHARLES W. CHAPMAN, M.D., M.R.C.P.,

Senior Physician to the National Hospital for Diseases of the Heart, Soho Square, W.; late Physician to the Farringdon General Dispensary, E.C.

It will be readily conceded that the value of a remedy is in proportion to its appropriateness for the individual case in which it has been prescribed. I want now to point out (*a*) some of the instances in which tonics may be misapplied, and (*b*) to show the importance of a correct judgment in the selection of an appropriate tonic when one is needed.

When a patient has so far recovered from a severe illness that a condition of debility only remains, what are the factors that make for a more or less complete restoration to health? They are mainly (1) the vis medicatrix naturæ, (2) a more liberal diet, (3) change of air and scene, (4) medicinal and other tonics. When normal health and strength have been obtained it is generally impossible to say to which of these means the greatest credit is due. Medical men would vary in their opinions. One would state that there is a natural tendency towards health which will in most cases assert itself when disturbing influences have been removed, provided this process is not thwarted by undue interference on our part. Another will affirm that improved diet has been the most potent element in imparting renewed vigour to the patient. A third will give the first place to the increased oxidation consequent upon a stay at the seaside. While a fourth, if he is biassed in favour of drugs, will claim for his tonic medicines the largest share in the beneficial result. The truth is, all four means have done good.

It has been my conviction for many years that no class of remedies has been more abused than those classed as tonics. When I was a pupil at the Royal General Dispensary, Bartholomew Close, "Mistura Quinæ" was with one of the physicians almost his only prescription. On his days it was customary to prepare several gallons of this mixture. Again, the expenditure for quinine at Guy's Hospital had grown to such an extent in 1873 that the treasurer expressed a wish that the use of this drug should be limited to cases where it was really necessary. Through the courtesy of the clerk of Guy's Hospital I am able to furnish the exact figures. It will be noticed there is a marked diminution in the quinine bill in 1874, the year following the treasurer's note:—1864, £761; 1865, £464; 1866, £567; 1867, £482; 1868, £745; 1869, £336; 1870, £512; 1871, £553; 1872, £704; 1873, £877; 1874, £187; 1875, £203; 1876, £200; 1877, £187. The lowness of the figures for four years following the initial reduction shows that the medical staff saw no reason to return to the use of quinine as an ordinary tonic.

I propose to discuss the use of tonics in general debility and in diseases of the more important organs.

In general debility after a severe illness a tonic is, in most cases, required; but the selection of the most suitable member or members of this class of remedies is of primary importance. Thus a patient recovering from an inflammatory affection of the digestive tract will do better on a vegetable bitter than with an astringent form of iron. The former would tend to improve the digestion, while the latter would be liable to cause irritation to the enfeebled mucous membrane and lead to constipation later on. In children who are recovering from broncho-pneumonia small doses of arsenic will be very beneficial. In all stages of syphilis, nux vomica or strychnine is the most suitable tonic with which to combine the more active and specific medicines. These instances will suffice for illustration.

In cases of want of strength, popularly called "debility," the public for the most part adopt one or more of three courses—take quack medicines, increase the amount of their stimulant, or seek professional advice. The mischief done by dosing with advertised tonics is writ large in the domestic treatment of delicate children. It falls to the lot of most practitioners to see children who have had some preparation of the hypophosphites alleged to contain every requisite for "the building up" of bone, muscle, and nerve, with the result that the unfortunate victim is in a weaker and more emaciated condition than when the much-vaunted medicine was commenced. Often matters have been made worse by addition of cod-liver oil with the idea of improving the nutrition. These patients have generally a muddy complexion, teeth stained by iron, constipation with offensive stools, a dirty tongue, and foul breath. We are

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told the child has no appetite, that it is fretful, sleeps badly, and frequently complains of headache. The mother will perhaps say her child has but recently returned from a prolonged stay at the seaside, or some inland health resort, and yet it has become worse rather than better for the visit. Cautious inquiry will, in most cases, discover that the patient has been improperly or excessively fed, to the extent possibly of dining late in the evening. Moreover such a life of excitement leads to wakeful or disturbed nights. Small wonder that a child living so unnatural a life should become feeble and peevish as a result of chronic catarrh of the digestive tract leading to malnutrition and auto-intoxication.

The obvious treatment of such a case is to stop all tonics and cod-liver oil, to prescribe simple and regular meals, allow no food or sweets to be taken between meals, insist on regular exercise in the open air whenever the weather is at all good, and see that a sufficient amount of sleep is obtained. This all seems apparent enough, yet how many children drag on a miserable existence for want of such treatment!

The medicinal treatment should consist of one or more doses of calomel, followed by rhubarb and soda, until the tongue has been cleaned and a healthy appetite obtained, after which a suitable tonic may be ordered, though, generally speaking, none will be required.

To another class of debility cases belong young men from sixteen to nineteen years of age, whose pallor is put down to over-work of some kind. Many of these patients have been victimised by quacks, and have gone through the gamut of advertised tonics with no beneficial result. We may discover we have to deal with early phthisis, diabetes, albuminuria, or some other serious disease, in which case valuable time has been lost. But the cases I am thinking of are those of lads who are suffering from the mental and physical effects of bad habits learnt at school, the former being caused or aggravated by reading pseudo-medical books and the terrifying remarks of well-meaning but injudicious friends. To give such patients iron or, still worse, strychnine, is to further stimulate their already over-sensitive sexual organs. What is needed is not an ordinary tonic, but treatment of the physical and mental conditions underlying the debility. One or more of the following points may require attention:—A tight or adherent prepuce, constipation, threadworms in the rectum, errors in diet, especially as to late and heavy meals and the use of alcohol, sedentary habits, etc. On the mental side the medical man should seek the confidence of his patient and be sympathetic in his manner towards him, and he must be assured that he will get ultimately quite well. As for drugs, bromide of ammonium with small doses of arsenic will be found to be of value.

I will now refer to habitual constipation as a cause of debility. Young women come before us complaining of great weakness whose condition may be in a great measure, if not entirely, due to retained fæcal matter with self-poisoning therefrom. If there exists deficiency or suppression of the menstrual function, the temptation in a very busy practice is to prescribe iron forthwith. Now apart from anæmia being possibly due to phthisis, gastric ulcer, renal or uterine disease, piles, etc., the condition may be entirely caused by chronic constipation. There may be a daily evacuation or even occasional attacks of looseness, and yet a large amount of old material still remain undisturbed. To give these patients iron without previously clearing the bowels is only to increase the mischief. And it must be remembered that aperients will have to accompany any ferruginous tonic that may subsequently be required. I have, however, seen the anæmia disappear and a clear, healthy complexion follow the use of aperients alone.

Another class of cases of skin pallor which refuses to respond to routine tonic treatment is that where the anæmia and general want of strength are consequent upon tight lacing. The effects of compression of the lower part of the chest and the upper part of the abdomen upon the contained organs are: (*a*) interference with the circulation, (*b*) lessening of their functional activity, (*c*) displacement of the liver and right kidney downwards. Among the results that might be expected to follow are pallor tending to become of an earthy character, palpitation, shortness of breath, indigestion and constipation, and disturbance of the menstrual function. It is surely folly to expect any benefit from tonics, or, indeed, any medicinal treatment, unless the pressure referred to be removed or materially mitigated.

M. O—, a well-developed, thick-set, single woman æt. 20 years, came under my observation in the

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out-patient room. She complained of breathlessness on slight exertion, want of appetite, constipation, and irregular menstruation. There was no history of any previous illness. When the chest was uncovered it was at once apparent that the corset she was in the habit of wearing was very tight. Moreover the facial expression became less anxious when the chest had full liberty of expansion. My question as to tight lacing was, as usual, met with an emphatic negative. A comparative measurement of the lower part of the chest and the cuirass showed a difference of nearly four inches. Thorough examination failed to give evidence of visceral disease of any kind, and a provisional diagnosis of "tight lacing" was made. A placebo, with careful directions for taking the inoffensive medicine, was prescribed, and more roomy stays ordered. There was manifest improvement in a week, and after a short time recovery to health was complete without the administration of a single dose of iron.

A. K—, a lady æt. 40 years, consulted me early this year on account of the following symptoms :— Stabbing pains at the heart, giddiness, shortness of breath on exertion, pain at the back of the head, a sense of constriction of the chest, a "tumbling sensation at the heart" coming on frequently and without any known provocation, and flushing of the face after meals; the patient also complained that her hands died off on her going to sleep. There had been no previous illness of any consequence. On examination the thoracic organs were found to be normal; there was some splashing on abdominal palpation. The patient was generally fat, and weighed 13 st. 5 lbs., while her height was 5 ft. 4 in. The difference between the body measurement and the stays was five inches. The case was diagnosed as indigestion with some gastric dilatation, probably due to, or at least aggravated by, tight lacing. Treatment was in accordance with the diagnosis, and it was so far successful that by three weeks all the symptoms had left, and she expressed herself as feeling better than she had done for a long time. I venture to affirm that no treatment would have given such good results if the pressure of the stays had been allowed to remain.

Phthisis is another disease where tonics are at times given without the exercise of requisite discrimination. Indigestion is a frequent early symptom of this disease, and is liable to occur in all stages of the affection. Mild vegetable tonics with an alkali, such as gentian and soda, is of more benefit than the more potent ones. This was my experience when working at the North London Hospital for Consumption a few years since.

Cardiac tonics are among the most misapplied. The routine prescription of digitalis when and wherever a murmur is discovered is in many cases not only unnecessary but positively harmful.

A. H—, a stout married woman about forty years of age, was seen in consultation. She complained of frequent severe attacks of palpitation followed by faintness, which was at times of an alarming character, especially during the night. There was also giddiness and a sense of "shaking" at the heart. On examination the heart's apex was at the fifth space half an inch to the right of the nipple line; the cardiac dulness was not increased in any direction; the action was excited, rapid, and regular; and, but for an occasional whiff systolic in time at the aortic orifice, the sounds were quite normal. The liver and lungs were healthy, and the urine normal. The patient had had cardiac tonics for some months, but she became worse rather than better by their use. The case was diagnosed as neurotic, and a favourable prognosis given. Treatment based on the diagnosis was followed by subsidence of all the heart symptoms and a general improvement in health.

Here was a patient with severe subjective cardiac symptoms of long duration who also had a suspicion of aortic stenosis, and although these symptoms were of long duration no secondary changes in the heart or other viscera were present. Again, cardiac remedies aggravated rather than relieved the symptoms. Instances of the misuse of direct cardiac tonics might be multiplied indefinitely.

Neuralgia, it may be urged, should at any rate be treated by tonics, especially by quinine. I agree that some neuralgias are very successfully treated with quinine. Take, however, neuralgia connected with a carious tooth. I think we have all been at times disappointed at the effect of quinine even in large doses, and the patient has had to go through all the miseries of an alveolar abscess. Pain in a decayed tooth is provoked by exposure to cold, irritating matter applied direct to the exposed

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nerve, or from poisonous material circulating in the blood due to mal-assimilation of food or constipation. The latter cause is illustrated by the following case:—An old friend sought my advice for neuralgic pains in the face which had refused to yield to treatment. On examination a tender and carious tooth was discovered. The tooth was too valuable to be removed, and it was quite one for conservative dentistry. The bowels were habitually costive, and the tonics which had been taken had increased the constipation. A dose of calomel was ordered, to be followed after four hours by a saline aperient which was to be repeated at intervals until fæcal masses were no longer to be seen in the evacuations. The following day my friend was quite well, and the tooth no longer painful even when tapped smartly. The tooth was subsequently stopped.

It is possible that increased vascular tension from the absorption of toxins from the bowel affected the exposed nerve-pulp, but, whatever may be the true explanation, the fact remains that the pain was removed by purging.

Tonsillitis used to be treated by large doses of Liq. Ferri Perchlor. under the belief that the disease was an outcome of debility, and that a tonic was required. Acting upon this theory, port wine and strong beef-tea were ordered. Under this *régime* suppuration generally occurred, with all the attendant suffering. The recognition of rheumatism as the chief etiological factor in this disease has led to the employment of the salicylates, but even here the efficacy of the drug will be more assured if it is preceded by a brisk purge.

No discussion on tonics can be complete without reference to alcohol. That alcohol is at times a valuable therapeutic agent no unprejudiced person will deny; whether it is a tonic in the usual acceptation of the term may be open to doubt. It is certain that with increasing knowledge of disease and the effects of remedies we have learnt to depend less upon alcohol than formerly. Alcohol is a useful cardiac stimulant, and has the power of increasing functional activity, as seen in some cases of atonic dyspepsia, and so indirectly assisting in the restoration to health. But this is a very different thing to the popular conception of the effect of alcohol even within the limits indicated. It may be necessary, even at the risk of doing harm elsewhere, to stimulate an enfeebled organ to activity, but the temporary good effect thus produced offers no warrant for the use of alcohol as a routine tonic.

The most mischievous of advertised "tonics" are the coca, iron, meat, and malt wines. That these preparations initiate and foster alcoholic excess, especially in women, I have no doubt. I would urge most strongly that the use of these medicated wines should be discouraged by the profession.

Akin to drugged wines are drugged foods. These preparations are extensively advertised, and have quite a literature of their own. We have predigested foods with malt and drugs of all sorts. These compounds have one characteristic in common in being more or less nasty; some of the meat "peptones" have a distinctly fæcal odour. Now I am far from saying that some of the predigested foods have not their legitimate use; but I am persuaded that we need more of the kitchen and less of the laboratory in the diet of the sick and convalescent. It seems to be taken for granted by some that the powers of digestion are entirely in abeyance during illness and convalescence, and that the digestive organs are to be encouraged in their apathy.

The following case illustrates the futility of trusting entirely to pre-digested foods. L. C—, æt. 58, was seen in consultation. She had been ill for some time from influenza, or something very much like that disease. The patient was neurotic, and had been for several days unable to retain nourishment. Malt wine, somatose, and several other preparations had been taken, but still the patient was alarmingly feeble. On inquiry I found the bowels had not been open for several days, and that although nutrient enemata had been given the rectum had not been washed out. The tongue was foul, breath offensive, and there was an icteric tinge in the conjunctivæ. There was nausea with a constant feeling of sickness. It was evident the patient would soon sink from sheer inanition unless more nutriment could be absorbed. The treatment adopted was to thoroughly clean the rectum with a turpentine enema, followed by small but frequent injections of beef-tea, yolk of egg, and brandy; calomel in $\frac{1}{10}$ gr. doses every fifteen minutes until the bowels were fairly relieved; and small quantities of milk and lime water with drachm doses of brandy were taken by the mouth as fre-

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quently as possible. The condition of the patient improved in a few hours, and the quality and quantity of the food was cautiously increased up to an ordinary diet.

Three points of practical value arise from study of this case :—(1) That it is useless to prescribe pre-digested foods because other forms of nourishment are not retained when foul material, even in small quantity, is allowed to remain in the intestine; (2) that nutrient enemata are of no avail unless the rectum is washed out at regular intervals; (3) that pre-digested foods should not be depended upon for nutrition until every effort to restore or improve the natural functions of digestion and assimilation have been made without success.

When a patient has been taking but little food it is sometimes thought the bowels may be left unrelieved for an indefinite time, and unsuspected auto-intoxication results. The small doses of calomel acted not only as an aperient, but rendered the bowels less septic, while the turpentine enema had a similar but more immediate effect upon the rectum. By these means the digestive tract was rendered able to deal with the lighter forms of invalids' food and trained up to make use of ordinary diet. The only other drugs used were bismuth, bicarbonate of soda, and nux vomica.

Two points should always be borne in mind in the selection of a tonic—(1) the nature of the illness and the special lesions known or suspected to have been present; (2) that the tonic will in no way be injurious to the natural functions of the body, the integrity of which must be maintained if convalescence is to proceed to complete restoration of health.

The possibility of idiosyncrasies in the matter of toleration of some tonic drugs should be remembered, and a patient's statement that certain drugs always disagree with him should not be lightly brushed aside. Some people cannot take quinine without severe headache following; others have urticaria after taking the drug; iron is not always tolerated, and strychnine, even in small doses, affects some patients with toxic effects.

Local Treatment of Exfoliating Glossitis.—A spoonful of Van Swieten's solution is taken in the mouth, and the head moved around until the tongue is thoroughly bathed in the fluid, which is then ejected. Gallois had his patients do this every day, and they were cured in a week.—*Journ. A. M. A.*, June 20th.

ON THE OPERATIVE TREATMENT OF FRACTURES.

Delivered at Guy's Hospital, May, 1903.

By W. ARBUTHNOT LANE, M.S.,

Surgeon to Guy's Hospital and to the Hospital for Sick Children.

(*Concluded from p.* 201.)

FRACTURES of the upper portions of the ulna and radius, occasionally complicating dislocation of the elbow-joint, require very careful treatment. Those of the olecranon, if seen before any change has taken place in the broken surfaces of the fragments, usually present no difficulty. They are fitted accurately together, are then drilled, and a screw is introduced. This is driven home till the olecranon fits accurately and is perfectly immobile. The head of the screw should be buried deeply. This can be ensured by using a burr to cut a concavity to occupy the head after the drilling is complete. If the head is not buried effectively it may after a long period cause irritation if it is pressed upon, and have to be removed. This, however, can be done easily through a very small incision, hardly greater than the breadth of the head of the screw. Fractures which you will find very difficult to deal with are those of the upper extremities of the radius and ulna. For instance, a fracture of the coronoid process complicating a dislocation backwards of the elbow-joint, if left alone after the dislocation has been reduced, may cause permanent incapacity of the arm and render it necessary to give up certain occupations, such as the navy or army, etc. Fig. 22 illustrates such a case. Till the displaced mass of bone had been removed the elbow could not be used. Its removal left a perfectly useful joint. If recognised at the time the fragment may be readily fastened in position by means of a screw which enters its lower aspect. This surface can be readily exposed by an anterior vertical incision over it.

The presence of a fracture of the upper end of the radius in a case of backward dislocation of the elbow may render it impossible to reduce the dislocation by manipulation, because of the upper fragment of the radius getting in the way. In such cases I have exposed the joint by a long posterior vertical incision, and after examining the condition

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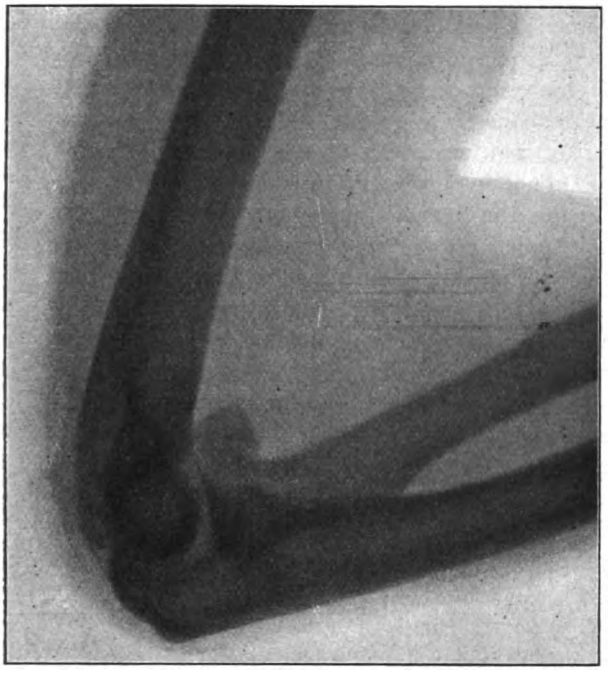
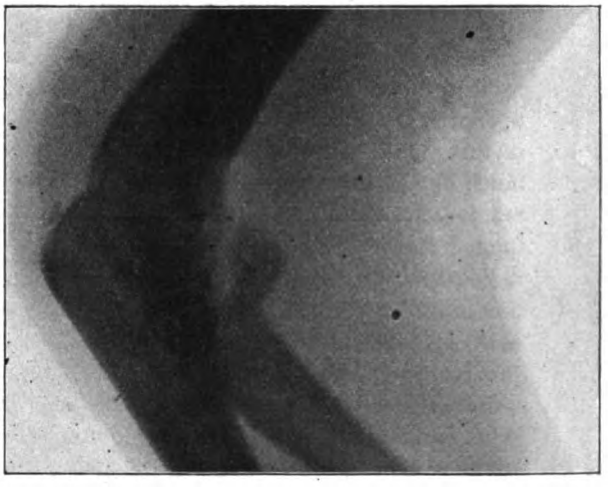
of the radius, either wiring on the fragment or removing it, should it consist, as it often does, of

FIG. 22.



the head and neck of the bone. After this the dislocation can be reduced, and a useful arm is obtained. Fracture of the upper end of the radius is not uncommon without dislocation of the elbow. In some cases the head is split vertically or obliquely, as in Fig. 23, in which I obtained a thoroughly efficient arm, with good range of movement, by wiring on the fragment. In others, again, as in Fig. 24, where the greater part of the head had been broken off, I got quite as good a result by excising the remains of the head and the upper part of the neck. You must be guided by the peculiar character of each particular case, and do the best you can under the circumstances. Any irregularity of the articular surfaces must of necessity be avoided. If you can fit the radial head on securely and accurately, do so by all means; but if not, I think you will obtain a more useful arm by cutting away the whole of the head and sufficient of the neck to allow of the development of a new structure in its place. The difficulty of screwing or wiring on of fragments of the upper part of the radius is made great by the presence of a number of large vessels and nerves in this situation. If you are in doubt as to their locality, look for them, and having defined their position, proceed carefully and thoroughly with your operation, without fear of doing serious damage to the patient. Operative measures may be called for if a perfect result is required in fractures of the shaft of the radius and ulna, or of either bone in which, from the presence of comminution or extensive swelling of the soft parts, or to any other cause, accurate apposition cannot be effected. I have seen quite a considerable number of cases of fractures of one or both bones of the forearm, in which I have been asked to operate some time after the receipt of the injury because of a more or less incomplete incapacity to fulfil the duties of their occupation. I will willingly confess that, as in the case of separation of the lower epiphysis of the humerus, much of this disability is due to a want of knowledge of the mechanics of bones and the mode of production of fractures and of the means that can be adopted for their improvement. The angular splints in common use, and especially the internal variety, are very largely responsible for this. The last thought of the anatomist is the consideration and the correct teaching of the mechanics of the skeleton, and unless the surgeon has some knowledge of this his treatment of fractures cannot be satisfactory. I have referred to this already fairly fully in the 'Clinical Journal,'

FIG. 23.



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entitled "Fractures of the Radius and Ulna" (vol. xx, p. 171). Owing to the fact that the ties in the length of the long bones of the forearm—or,

FIG. 24.

in other words, the surrounding soft parts—are much less bulky, less readily damaged, and are therefore not so shortened up by the extravasation of blood and subsequent inflammation as in the case of the long bones of the leg, the surgeon does not experience the same great and often insuperable obstacles to effecting accurate apposition of fragments in the upper as he does in the lower extremity. Still in spite of this you will come across cases of fracture of the radius and ulna in which you will not be able to restore these bones to their normal form by any means other than operative. Fig. 25 illustrates an extreme instance of this. The fragments were exposed, and with much difficulty were fitted accurately together, and retained in this position by wire loops which perforated them. A superficial examination of the radiograph will convince you at once that restoration of the form and function of these bones could not have been effected by any manipulative process with which you are familiar.

In performing these operations make long incisions, carefully avoiding any important nerve and vessel. The operations are much more simple than those on the bones of the leg, and apposition is effected with comparative ease. The fragments are retained in position with wire, since they rarely afford a suitable grip for a screw. As the fractures are usually on the same level, and the grip of the wire is less rigid than that of a screw, there is more play between the fragments, and the restoration of the form of the bone is frequently not quite as perfect as it is in fracture of the tibia and fibula. Still, in recent fractures, by an operation you can always count on restoring completely the functions of the bones, whose shape is almost, if not quite, as perfect as it was before the accident.

Do not employ internal angular splints in putting up the arm after operating, but place the arm in the supine position, using anterior and posterior splints, including the hand as far as the metacarpo-phalangeal joints. While considering the difficulty in restoring perfectly accurately the axes of the fragments of the radius and ulna when the line of fracture is transverse, or nearly so, owing to the slight wobble on the wire loop or loops, it is well to remember that in the young subject the new material, commonly called callus, which is thrown out is not limited to the seat of the fracture, but extends along the whole length of the shaft. It is

FIG. 25.

by means of this new material that an entirely new shaft is modelled if the lines of force do not correspond to the residual fragments.

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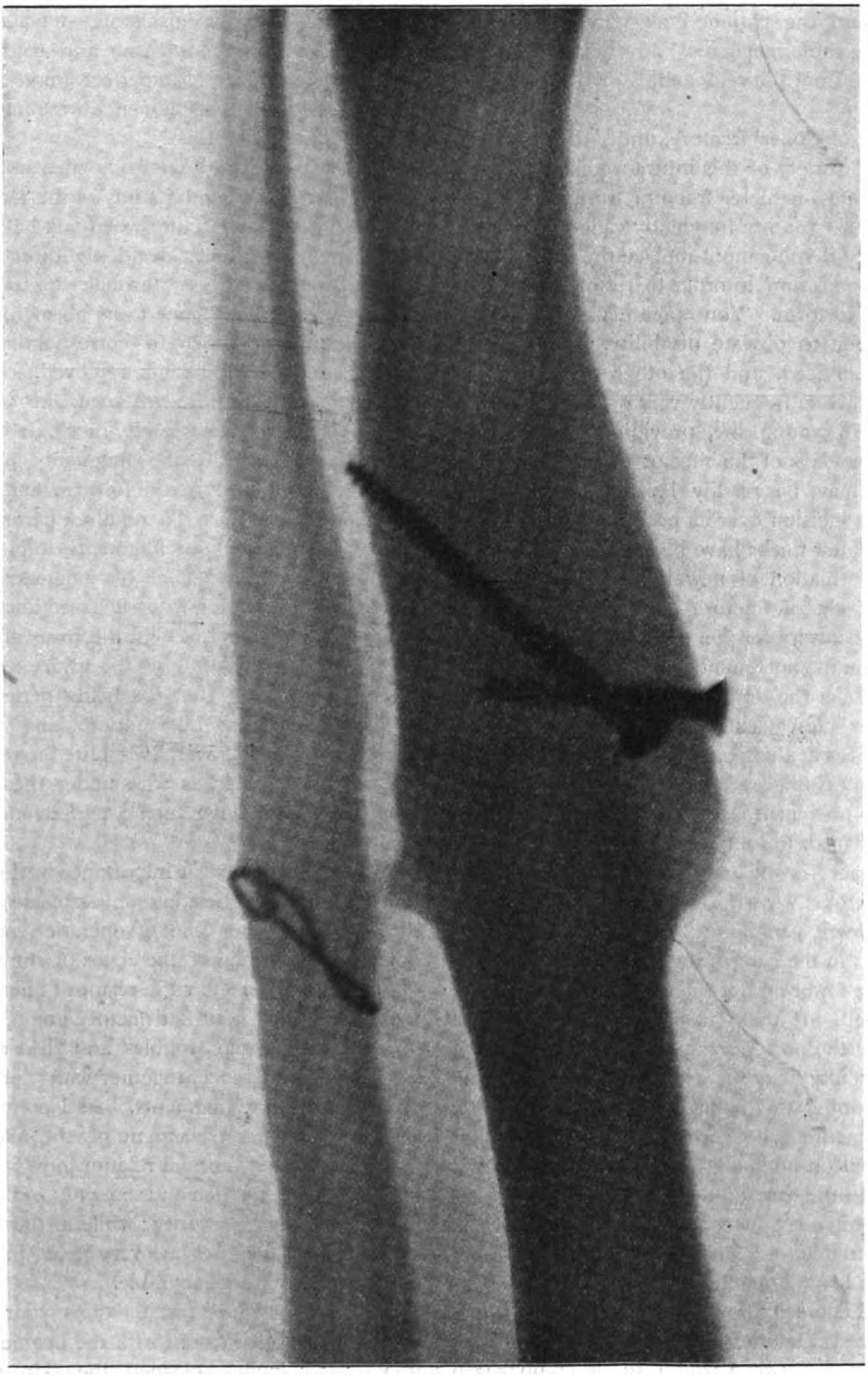
FIG. 26.



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These radiographs show this very well. Later in adult life the callus is limited to the seat of fracture, and the patient does not derive subsequently the same mechanical advantages as ensue in early life from the remodelling of the bones of the part.

In cases of Colles' fracture, under which we will include the variety of this injury which takes place in young life—namely, fracture through the epiphysial line,—you are frequently called on to interfere actively if you cannot replace the fragments in accurate apposition in order to restore the radius to its normal form. You operate usually for two reasons—one to obviate disability, which may be very great indeed, and the other to prevent deformity, which is frequently very distressing to the sufferer. In young life, providing you interfere within a few days of the receipt of the injury, the epiphysis may be readily levered into position through an incision over its posterior surface.

If a very few weeks have been allowed to elapse, the rapid formation of new bone which develops about the seat of fracture in this class of injury makes it necessary for you to cut or chisel through the junction in such suitable plane as will enable you to place the lower epiphysis in a position which is as perfect as you can obtain under the circumstances at this period. Having done this, it is almost always necessary, in order to retain it in position to prevent it returning to that into which it was displaced, to join the epiphysis to the shaft by a fine tack, or by one which you can readily fashion out of silver wire. In vigorous adult life the lower fragment can be exposed and levered into position, as in the case of the epiphysis. In feeble old age the lower end of the radius breaks up like an egg-shell. If there is much destruction and crushing of the posterior surface of the fragments, much difficulty may be experienced in securing and in retaining the bones in perfect coaptation. It may sometimes be necessary to make another vertical incision immediately external to the radial vessels. In the case of the old subject in whom the lower fragment is comminuted and the bone thin and friable, it is impossible to effect perfect restoration by any operative measures.

You will come across two classes of cases which have resulted from the unsatisfactory treatment of fractures, and between which there is no sharp limitation. They are due either to the union of the fragments in such a position of mutual displacement as renders it impossible for the sufferer to perform his normal mechanics sufficiently freely or painlessly to earn his living and enjoy his life, or to such condition of imperfect union or absence of union between the fragments bringing about the same or similar results.

The treatment is practically identical in both cases. From the careful study of the radiographs, and of the seat of fracture when you have exposed it thoroughly, you must decide the direction of the planes in which to saw through the fragments in order to restore each bone to its normal form, with only diminution in length corresponding to the portions of the fragments removed. Except in the comparatively rare instances in which the section is made transversely, in single bones it is necessary to divide the fragments along two separate planes, and in the forearm and leg along four distinct planes. To obtain a perfect restoration of the axes of the fragments to one another may require a good deal of judgment or skill. The means of dealing with such sectional surfaces resemble those employed in the cases of fractures operated on at the time of the injury, except that the surfaces cut by the saw, being perfectly plane and approximating but not locking into one another, a screw is generally required to retain them immovably together, as wire under these circumstances does not give such a perfect and firm coaptation.

The difficulty next in importance in these operations is that of obviating any excessive hæmorrhage, both at the time of operation and subsequent to the closure of the edges of the wound.

Figs. 26 and 27 show the result of operations for these conditions of unsatisfactory union, and they give an idea of the troubles and risks associated with such interference, together with the methods of fixation of the fragments. As I pointed out to you before, the replacement of the axes of the fragments in their normal relationships will make a very considerable demand on your skill, even in cases of moderate severity; while in the worst sort of case the difficulties are very great indeed, and occasionally quite insuperable.

In treating compound fractures your methods must vary to some extent with the degree of infection. After having cleaned the skin your first endeavour must be to remove any organisms that

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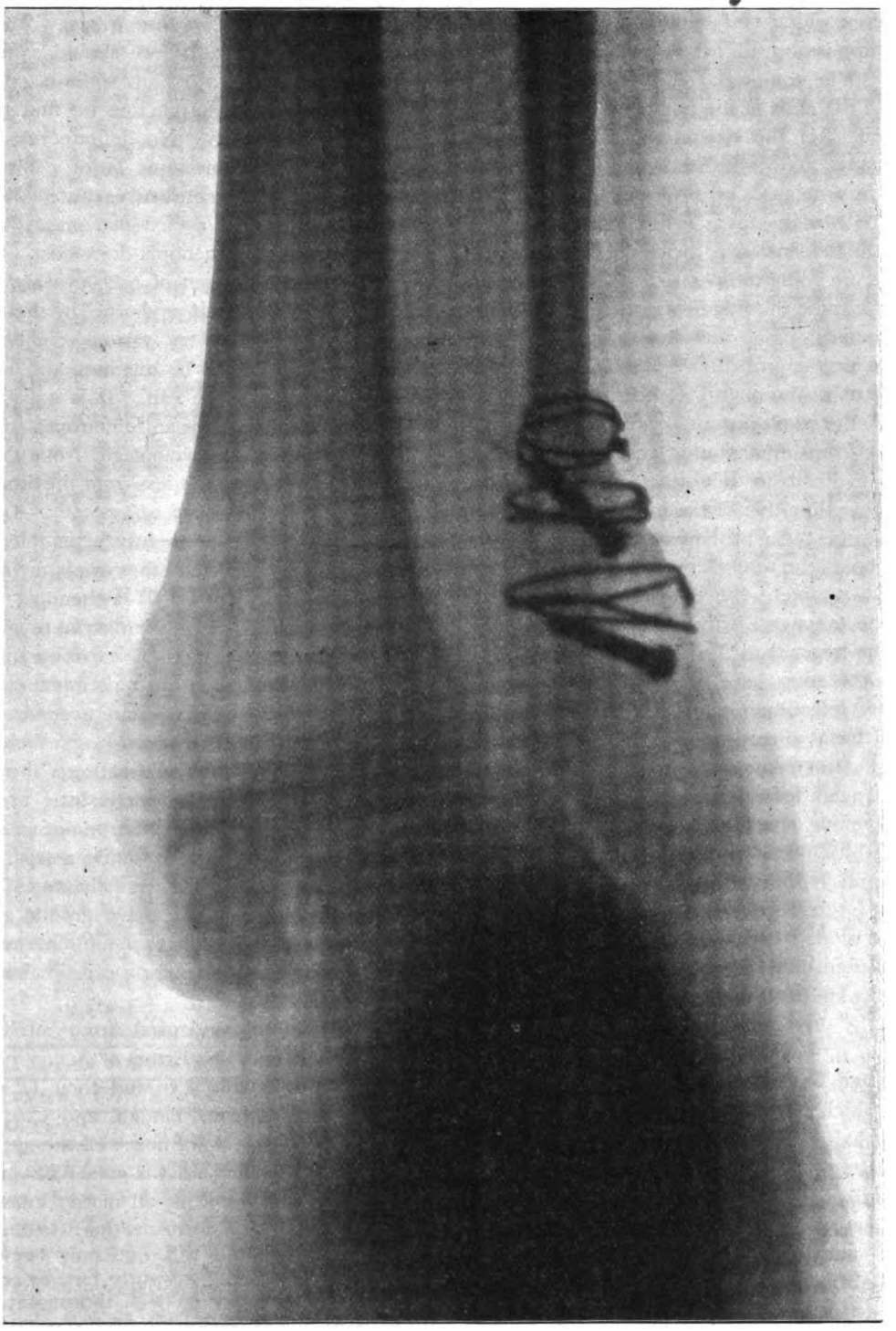
FIG. 27.



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may have effected an entry into the wound, for the reason that if the wound is not rendered sterile the wire or screw which you employ to retain the fragments in apposition will act as foreign bodies, and will have to be removed. A variable amount of necrosis of the ends of the fragments will probably result, and after the separation of the sequestra the fragments may unite at a bad angle, or union may be very delayed, or a fibrous junction may result. To obviate these evil consequences you must, in the first instance, incise the limb as freely as if you were going to operate in a case of simple fracture. Expose the fragments freely, and if there is any doubtful tissue anywhere, excise it freely. If the infection is probably very trivial, after drying the soft parts as thoroughly as possible with sterile pads, pack the whole interior with sterile cyanide gauze free from any irritating germicidal solution, which is inclined to inhibit the vitality of the tissues rather than that of the organism. If on the removal of the gauze at the end of twenty-four or thirty-six hours the wound appears dry and healthy, you might perforate the bones and connect the ends of the fragments with wire. Make the holes in the bone larger than the calibre of the wire, so that after the edges have been accurately coapted, should any infection remain in their interior the holes will form a ready means of exit. Having secured the fragments firmly in apposition, pack the wound again for another period of one or two days. Remove this packing, and if the parts appear healthy, bring the limits of the skin wound together and reduce the packing. Repeat this process at intervals till you feel justified in closing the wound along its entire length. If there is any gaping, Robinson's decalcified cancellous tissue will serve as an ideal dressing.

Prolonged Lactation.—Nussbaum reports two cases in which lactation was prolonged for from one and a half to two years after weaning, although repeated attempts had been made to suppress the flow of milk. The patients were suffering from severe anæmia. When the cases came under the care of the author they were at once placed upon 0·5 gr. of antipyrin four times a day, together with mild cathartics. A breast binder was also applied. This treatment was soon followed by a complete cessation of lactation.—*Medical Record*, June 20th.

Anisometropia.—There are three varieties of anisometropia:

1. *Simultaneous Binocular Vision.*—The treatment varies considerably; we should expect that each eye should be fitted up with its correcting glass in all cases, but in practice we find this will not suit many patients. No definite rule can be laid down. If the ametropia be of a low degree, and binocular vision acute and easy at all distances, and if there be no asthenopia, glasses are not necessary. When asthenopia does exist, place the proper correction in front of each eye and try the binocular effect; if this is borne by the patient, order these; if not, try various combinations. Full correction should be our aim, and we must get as near to it as we can. It is a good plan, when ordering glasses for anisometropia, to deduct a little more for the cycloplegic from the most ametropic eye and a little less from the other.

2. *The Eyes are Used Alternately.*—One eye may be emmetropic or slightly hyperopic, and is used for distance, and the other eye is myopic, and is used for near work. If asthenopia be not present the patient will not thank you to give him any glasses. When asthenopia exists we shall find, probably, that both eyes are ametropic, one more so than the other, and glasses are needed.

3. *One Eye is Permanently Excluded from Vision.*—When the difference between the eyes is great, the most defective eye is little used, and tends to become amblyopic, if it be not so already. In such cases it is advisable to give a separate pair of glasses correcting this eye fully, and when the other eye is emmetropic to put a ground glass on this side, and the patient should be made to practise with these glasses for a certain time every day.

The above is condensed from Mr. Ernest Clarke's book on 'The Errors of Accommodation and Refraction of the Eye and their Treatment,' published by Baillière, Tindall, and Cox, and it affords a good example of how well the author has succeeded in retaining what is essentially practical, at the same time omitting all matter unnecessary for the busy practitioner or overburdened student. Mr. Clarke's volume will not only be found a reliable guide in practical work, but, by reason of the subject matter having been thoroughly written up to date, it will also be useful as a storehouse of information, and as an intelligible means of instruction.

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THE CLINICAL JOURNAL,

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A LECTURE ON SOME FORMS OF OPHTHALMIA.

Delivered at the Medical Graduates' College and Polyclinic, June 10th, 1903.

By E. TREACHER COLLINS, F.R.C.S.,

Surgeon, Royal London Ophthalmic Hospital; Ophthalmic Surgeon, Charing Cross Hospital.

"IF preventable, why not prevented?" is a question which has been mostly applied with reference to consumption, but it is one which might be asked with equal aptness concerning ophthalmia. Several different forms of inflammation of the conjunctiva are known to be connected with the growth of certain micro-organisms in the conjunctival sac. These, and some others in connection with which as yet no definite micro-organisms have been isolated, are contagious, and are spread by the transmission of discharge directly or indirectly from one person to another.

The micro-organisms, pure cultures of which have been shown to be capable of producing conjunctival inflammation, are readily killed by drying, and do not form spores. It is improbable, therefore, that dried secretion, if converted into dust, would prove infective, and what may be spoken of as aërial infection seems unlikely.

A hyperæmic condition of the conjunctiva, excited by exposure to dust, wind, or in other ways, may predispose the conjunctiva to infection by rendering it a more suitable soil for the growth of micro-organisms, or one that is less likely to resist their invasion.

We must, however, remember that a hyperæmic condition of the conjunctiva, on account of the irritation to which it gives rise, renders infection of an eye more likely to take place from fingers or handkerchiefs used to rub or wipe it.

We shall find it a good working hypothesis, though possibly not absolutely true, to regard all cases of ophthalmia as the result of some infective

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discharge which has gained entrance to the eye. This discharge need not necessarily come from the conjunctiva. It may have been produced by some other mucous surface, such as that of the genital organs.

In *ophthalmia neonatorum* the infection is derived from such a source. During birth, in the passage of the head through the vagina, any discharge from the lining membrane of that passage is liable to adhere to the skin of the eyelids, or to the eyelashes. Then, after the birth of the head, when the child first opens its eyes, that discharge may be carried into the conjunctival sac and cause infection. It is improbable that any of the vaginal secretion often gains entrance to the eyes during the passage of the head through the vagina, for the eyelids are then tightly closed, and it could only be so introduced by separation of them with the finger of the examining accoucheur or by an instrument.

If an irritant is introduced into the conjunctiva of any but an infant's eye, there is at once excited a flow of tears which mechanically tends to wash it away. The tears, moreover, have been shown to possess a weak bactericidal property. An infant at birth secretes no tears, so that its conjunctiva does not possess this flow of a bactericidal fluid as a means of defence. It becomes necessary for the surgeon to provide it with a substitute, so as to prevent infection. This is best effected by washing the eyelids with an antiseptic lotion and wiping away with absorbent cotton wool any discharge adherent to the eyelashes immediately after the head has been born, and afterwards introducing into each eye a drop of solution of a silver salt.

The systematic use of 2 per cent. nitrate of silver drops to the eyes of all new-born infants in lying-in establishments has practically abolished this disease from them. Credé, who introduced this treatment, has shown that out of 2266 cases before its employment 9·97 per cent. had ophthalmia neonatorum. After its employment, out of 1160 cases only 0·086 per cent. had their eyes affected. Kostling has collected the records of 17,000 births tabulated by thirteen observers, before the introduction of Credé's treatment. Nine per cent. had ophthalmia neonatorum. After Credé's treatment, 24,000 births, tabulated by thirty-one observers, showed only 0·65 per cent.

It might be thought that dropping a 2 per cent. solution of nitrate of silver into the eyes of new-born infants was likely to cause some irritation, but nothing more than a transitory redness is usually excited, which need hardly be taken into account when the immense prophylactic value which this measure affords is considered.

Protargol is a silver salt which gives rise to much less pain and irritation than nitrate of silver, and a 20 per cent. solution has been used by several observers with satisfactory results.

Ophthalmia neonatorum may be regarded, then, as a preventable disease; notwithstanding this, nearly a third of the inmates of blind asylums are found to have lost their sight from this affection. A large number of the cases in which it occurs are those whose mothers have been attended by midwives; and it would be a useful thing, now that midwives have to be registered, if compulsory notification of cases of ophthalmia neonatorum occurring in the practice of all persons attending women in labour, other than medical men, was enforced. For not only is ophthalmia neonatorum a preventable disease, but all cases may be cured without any affection of the sight if seen sufficiently early. I always hold that the worst stage of a case of ophthalmia neonatorum should be that at which the patient is first seen. As soon as appropriate treatment is adopted the condition should begin to mend. If the cornea is unaffected when the patient is first seen, the case should be cured without its becoming involved.

Appropriate treatment consists in the skilful application of a 2 per cent. solution of nitrate of silver once every twenty-four hours to the inner surface of the everted lids by the surgeon himself; and the careful washing away of all discharge every one or two hours by the mother or nurse with a zinc chloride lotion, one grain to the ounce, or perchloride of mercury lotion, one eighth of a grain to the ounce. The nitrate of silver lotion should not be applied more frequently than once in twenty-four hours; otherwise a membranous ophthalmia is liable to be produced.

In about two thirds of the cases of ophthalmia neonatorum the gonococcus has been found to be present in the discharge, and in about the same proportion of cases the history of a leucorrhœal vaginal discharge can be elicited from the mother. If infection occurs during labour, in the way I have just mentioned, the symptoms always begin before

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the fifth day. In cases which occur later, infection must have been brought about by contaminated water, towels, linen, sponges, etc., and is attributable to want of proper aseptic cleanliness on the part of the monthly nurse or mother.

Gonorrhœal ophthalmia in the adult is a much more severe affection, and not nearly so amenable to treatment as in the infant. Cases which we have to deal with from the very early stages will occasionally, in spite of all our efforts, get ulceration of the cornea, which may go on to perforation and destruction of the eye. Prevention of such an affection is therefore of the very greatest importance. The general public are, as a rule, very ignorant about the nature and danger of infection. It is therefore well to warn any patient suffering from gonorrhœa of the genital organs of the danger of getting discharge into the eye, and the importance of disinfecting contaminated linen or of quickly destroying any foul dressings.

There is a case recorded of a lad who had some slight inflammation of the margins of the eyelids; his mother, seeing this, advised him to employ the popular remedy of bathing his eyes with some of his urine. Unfortunately she did not know he was suffering from gonorrhœa, and a violent attack of gonorrhœal ophthalmia was excited.

I have known a house surgeon and a nurse get their eyes infected while treating cases of gonorrhœal ophthalmia, and I think it advisable, when any discharge is being syringed away from an eye, for those in attendance to put on protective glasses.

If a patient presents himself with only one eye affected, immediate steps should be taken to secure the safety of the other. The affected eye should at once be covered up. This is best done with what is known as a "Buller's shield," which consists of a watch-glass sewn into a piece of mackintosh. It is fixed with adhesive plaster above to the forehead and on the inner side to the nose, being left unattached at the outer and lower parts for ventilation. The safety of the fellow eye is thus ensured without the patient being kept in darkness, for he can with it see through the watch-glass.

In the early stages of gonorrhœal ophthalmia, when there is considerable swelling of the lids and conjunctiva without any discharge, strong caustic applications should not be employed. At this stage it is best to keep ice-pads constantly applied, by which means the amount of swelling will often be considerably reduced. These cases require constant attention, and it is desirable when possible to have a special nurse in attendance.

If there is much swelling of the ocular conjunctiva, so-called chemosis, and it looks as though the nutrition of the cornea was likely to suffer from constriction of its vascular supply, it is well to scarify the conjunctiva and let out some of the œdematous fluid.

As soon as the purulent discharge begins to form, applications of nitrate of silver 2 per cent. to the inner surface of the everted lid should be commenced, and continued once in every twenty-four hours, the discharge in the intervals being frequently washed away with a perchloride of mercury or zinc chloride lotion.

I have mentioned how a *membranous conjunctivitis* may be produced by the too frequent application of caustic solutions in cases of ophthalmia. A membranous ophthalmia of the conjunctiva may also be excited by an infusion of jequirity applied to the eye. In a burn of the conjunctiva from lime, a superficial slough forms which presents a membranous character. A membranous condition of the conjunctiva may then be excited by a variety of irritants. Where a case arises, however, spontaneously, apart from any local application to the eye, the suspicion of its being one of diphtheria should at once suggest itself.

It used to be thought that it was only the severer cases of membranous ophthalmia which were diphtheritic, those where there was, in addition to much hard brawny swelling, an actual slough in the superficial tissue, which on separation provoked bleeding. The cases in which the membrane consisted only of an exudate of fibrin were spoken of as croupous. We now know, that just as there are cases of diphtheria of the air-passages in which the local symptoms may be so mild, that they would have been thought nothing of, were it not that attention was directed to them by ensuing paralysis, so the local symptoms of diphtheria of the conjunctiva may be of a very mild description. It is, in fact, from the clinical appearances of the eye only, impossible to say whether a case of membranous ophthalmia is or is not diphtheritic. A bacteriological examination alone can determine it. It is of course just as important to notify a case of diphtheria of the conjunctiva as a case of

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diphtheria of other parts, and the bacteriological test should be applied to all cases.

True diphtheria of the conjunctiva is best treated by the employment of antitoxin serum. It is most satisfactory the way in which membrane can be got rid of by its means, when it has been found most resistant to the use of local antiseptic applications.

We have thus considered two forms of ophthalmia definitely due to micro-organisms; gonorrhœal ophthalmia due to the gonococcus, and diphtheritic ophthalmia due to the Klebs-Loeffler bacillus. I have now to refer to a third, what is usually spoken of in text-books as acute catarrhal ophthalmia, but which is better described as *acute muco-purulent ophthalmia*, and with which is frequently associated the Koch-Weeks bacillus, a specimen of which I have placed under the first microscope.

It is an affection which comes on rapidly, with much injection of both ocular and palpebral conjunctivæ. The injection of the ocular conjunctiva in places is often so intense as to make it look as though there had been a subconjunctival hæmorrhage. Magnification of these patches, however, shows them to be made up of distended capillaries. It is the bright red appearance of these patches which has led to the affection being popularly spoken of, especially on the other side of the Atlantic, as "pink eye." In this country it is more usually termed "the blight," probably from the highly contagious nature of the affection causing several members of a family to become affected at about the same time. It is accompanied by a yellowish muco-purulent discharge, which collects during the day at the inner canthus, and during the night causes the margins of the lids to become stuck together.

The same set of symptoms sometimes seems to be caused not by the Weeks bacillus, but by a diplococcus, which is encapsuled, and has all the appearance of the pneumococcus. This micro-organism is at times met with in the conjunctiva of perfectly healthy eyes, and it would seem that it is only when certain predisposing causes are present that it is capable of exciting inflammation.

The most important element in the treatment of a case of acute muco-purulent conjunctivitis is to keep the discharge well washed away. The disease runs a rapid course, and tends to get well of itself. Thorough washing with sterilised water would probably be as efficient a treatment as any. I generally prescribe a weak antiseptic lotion, such as perchloride of mercury 1 in 4000, and some boracic ointment to be applied to the margins of the lids at night.

Being highly infectious, it is important to warn those affected from allowing others to use the same towels, handkerchiefs, etc., as themselves. In the event of a case of this description occurring at a school or institution, the patient should be at once separated and kept isolated until all the discharge has ceased.

One of the commonest forms of ophthalmia in this country is what is best termed *angular conjunctivitis*. It is a chronic affection which, if untreated, lasts for months. The injection is chiefly of the palpebral conjunctiva, the ocular conjunctiva being little, if at all, involved. The most striking characteristic of the affection is the marked injection which it presents at the outer and inner canthus, both of the lid margins and of the subjacent skin. The discharge, which is not very great, is of a grey, sticky, mucoid character. At the two angles of the eye the lashes are often stuck down to the reddened skin by the sticky secretion. The affection is associated with a characteristic and easily recognised bacillus termed the diplo-bacillus of Morax, a specimen of which you will see under the second microscope. The most satisfactory feature of the affection is the rapid way in which it yields to treatment. Without treatment it drags on indefinitely. With sulphate of zinc drops, two grains to the ounce, applied three times a day, and boracic acid ointment applied to the lids, it rapidly gets well.

I come now to describe certain forms of ophthalmia which are accompanied by granulations on the surface of the conjunctiva. They might be spoken of as cases of *granular ophthalmia*, but if so, under that term must be included several distinctly different affections.

First of all, granulations or irregularities of the surface of the conjunctiva may be due to enlarged papillæ. These are mostly met with on the tarsal conjunctiva, especially at the line of its junction with the retro-tarsal fold, where normally there is a tendency to the formation of papillæ. Any form of inflammation of the conjunctiva which has become chronic is liable to be accompanied by

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some enlargement of the papillæ, so that the inner surface of the lids when everted presents a velvety appearance.

There is a peculiar form of affection of the conjunctiva, which disappears during the cold weather but comes back again in the spring. It is called *spring catarrh*. The most striking characteristic of it is the immense overgrowth of papillæ of the palpebral conjunctiva. The granulations thus produced present certain peculiar features, to which attention has to be directed in order that they may be differentiated from those which occur in trachoma. They have markedly flat tops, and their edges fit in against one another so as to present an appearance comparable to paving-stones. On the surface of them is a peculiar whitish haze, which is best compared to the bloom on the surface of a plum, and is attributable to thickening of the epithelium covering the papillæ. If grasped with forceps they will be found to be exceedingly tough, feeling like pieces of cartridge.

Some two years ago I was asked, together with another ophthalmic surgeon, to see a gentleman who had already consulted eight others with reference to his eyes. Four of them had told him he had trachoma, and four that he had spring catarrh. After hearing a history of his symptoms and carefully inspecting the inner surface of his lids, the other ophthalmic surgeon present said he considered the case one of trachoma. I, on the other hand, had arrived at the opinion that it was one of spring catarrh. So the poor gentleman was not much better off; he now had five opinions one way and five the other. To arrive at some more definite conclusion I excised a piece of the granulated surface from the retro-tarsal fold for microscopical examination. The sections showed enlarged papillæ composed of dense fibrous tissue, and no nodules of lymphoid tissue such as are met with in trachoma.

Besides the affection of the palpebral conjunctiva, there is, in some cases, a thickening of the ocular conjunctiva at the corneal margin, which is also of a papillary character. This new growth sometimes encroaches on to the cornea itself, restricting its circumference in an irregular manner. It is noteworthy that it is in just that portion of the ocular conjunctiva in which normally there is some down-growth of epithelium and a tendency to papillary formation that this pathological papillary formation takes place, and not elsewhere.

The symptom of the affection of which the patient makes most complaint is an intolerable itching or irritation about the eyes, and it is the relief of this to which treatment has to be directed, as so far nothing that will cure the disease has been discovered. It is commonest in children, and generally, after having relapsed regularly for three or four years, ceases spontaneously. The lotion which I find gives the most relief is one of carbolic acid 1 in 40. It has a mild local anæsthetic action, and it is best to use it quite cold or iced.

In the conjunctiva there is normally a certain amount of adenoid tissue, mostly situated in the retro-tarsal folds. In many children this adenoid tissue, like the adenoid tissue of other mucous surfaces, is liable to become increased. It then shows itself as little nodules or granulations of the conjunctiva. These nodules are never much larger than a pin's head. They are grouped together in little chains, and have no tendency to run together and become confluent. They are usually most marked in the lower *cul-de-sac*. The children in whom they are present are usually found to have enlargement of adenoid tissue in the air-passages, and a granular condition of the pharynx. The condition is frequently met with apart from inflammation, but as overgrowth of adenoid tissue in the air-passages renders them liable to attacks of catarrhal inflammation, so enlargement of follicles in the conjunctiva predisposes to inflammation of that membrane. When the two are associated, the condition is generally spoken of as *Follicular Conjunctivitis*. It is very necessary to be able to diagnose a case in which there is simple enlargement of normal adenoid tissue, from trachoma, where there is extensive new formation of lymphoid tissue in situations in which it is not normally present. In the early stages of these affections this may sometimes be difficult, but in their later stages the two are well defined. Follicular conjunctivitis never goes on to the formation of cicatricial tissue, which commonly occurs on the disappearance of the lymphoid tissue in trachoma. Hence in follicular conjunctivitis none of those sequelæ the result of cicatrisation, such as entropion or contraction of the conjunctival sac, occur. Vascularity of the cornea, the condition which is

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termed pannus, a common accompaniment of trachoma, is never met with in follicular conjunctivitis. The prognosis, therefore, of the two affections is very different, and distinction between them in their early stages is of great importance.

In *Trachoma* the characteristic granulations, which have been aptly compared in appearance to frog spawn or grains of boiled sago, are not confined to the retro-tarsal fold, but extend also to the tarsal conjunctiva. Unlike those of follicular conjunctivitis, they present many irregularities in size and shape.

I saw it stated in a medical journal a little while ago, that notwithstanding all the time and trouble which has been expended in the research as to the nature of trachoma, our methods of treatment of that affection had not improved; that sulphate of copper was known to be the best remedy in the time of Hippocrates, and is the best remedy still. In this there is a certain measure of truth. Sulphate of copper is probably the best local application we have, but of recent times it has become possible to considerably shorten the duration of treatment in cases of trachoma by the introduction of operative procedures.

By the operation of expression with either the roller forceps or with Grady's forceps, the whole of the follicular stage of the disease can be done away with. After the eye has been thoroughly cocainised, the conjunctiva, wherever the enlarged follicles are present, is grasped with the forceps, which are then gradually drawn forwards, squeezing out the lymphoid material. Local applications of sulphate of copper, nitrate of silver, or strong perchloride of mercury solution have subsequently to be employed; but granulations which would take months to become absorbed by their use only, are rapidly got rid of by means of the forceps without any subsequent undue cicatrisation.

The prevention of trachoma is a big and complicated subject, but one of so much importance to the community, both in this country and others, that I must briefly refer to what has so far been tried and effected. Trachoma is probably responsible for more loss of sight and suffering than any other eye affection. The numbers that suffer from it or its sequelæ in Eastern countries is enormous. From my own experience in Persia I should say that quite 50 per cent. of the population there have been affected. Morax states that in Alexandria it was quite the exception to find a native who, when the lids are everted, does not show signs of passive or active trachoma.

In this country the affection is confined almost entirely to the poorer classes. It is stated that it was introduced by the soldiers returning from Egypt during the Napoleonic war. It spread through the barracks, and then, on the discharge of the affected soldiers, they carried infection to other members of the community. It was the large increase of eye disease from this cause, just a hundred years ago, which led to the establishment of what is now known as the Royal London Ophthalmic Hospital, Moorfields. In connection with this it is pleasing to read the following statement made by Sir Thomas Longmore before the Royal Commission on the Blind in 1889.

"There is a record showing that on December 1st, in the year 1810, the number of soldiers on the Pension List for total blindness the result of ophthalmia was 2317. But for the last twenty years there has been no trace of epidemic ophthalmia in the British army. Better treatment and better sanitary arrangements have banished it from the army. Under ordinary conditions, and when it exceptionally did occur, as it did in Egypt in 1882, the improved treatment prevented it from being followed by any permanent bad consequences."

Though up to the present no micro-organism has been definitely ascertained to be the cause of trachoma, there can be little doubt that it is contagious. In London for a long time the pauper schools have been a hot-bed for its dissemination. In certain districts for some years past measures have been taken to isolate all the affected children, and to carry out their education and treatment in a separate building. The most satisfactory results have thus been obtained, and now the Metropolitan Asylums Board are undertaking to extend the system to all the pauper schools of London.

I was asked by one of the members of that Board a short time ago, whether by these means it is likely that in some years the disease will be stamped out in this country. My reply was, "Not as long as fresh cases are continually being introduced amongst the aliens who are constantly flocking to this city." Many of these, as is well known, are Polish Jews, who seem peculiarly liable to be affected.

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In the United States the danger of allowing the introduction of this affection by immigrants has been recognised. In 1897 the Treasury Department of Washington declared trachoma to be a dangerous contagious disease, thus putting immigrants so afflicted clearly within a class of aliens prohibited from entering the country. The result of this law during the first three years was to exclude 900 cases from the country which were likely to form fresh foci for the spread of the disease. There is evidence to show that in the Eastern States there has of recent years been a considerable diminution in the amount of trachoma, attributable in part to this cause, though possibly also in part due to improved methods tending to shorten the course of the affection.

This exclusion of affected aliens from the United States tends probably to increase the number in this country. For at ophthalmic hospitals it is now not uncommon to meet with patients applying for treatment for trachoma before proceeding on their way to America, or after being refused admission there.

A DEMONSTRATION OF CLINICAL CASES.

Given at Charing Cross Hospital,

By HERBERT F. WATERHOUSE, F.R.C.S.,

Senior Assistant Surgeon, Charing Cross Hospital.

(*Concluded from p.* 160.)

Case 5.—Unilateral Excision of the Tongue for Epithelioma.

The next case I want to show you is one of cancer of the tongue. The result is a fairly favourable one, because it is just about two years since the patient was operated upon. The patient is a man forty-six years of age, who, two years ago last May, was placed under my care by Dr. Victor Sapp with a large epithelioma involving an inch of the margin of the right half of the tongue, and some distinct glandular enlargement in the right submaxillary region. There was no question about the diagnosis; it was obviously an epithelioma, and it had been noticed by the patient three months before he sought any advice. The neoplasm was distinctly limited to the right half of the tongue; it did not go anywhere near the middle line, and knowing that recurrence very rarely occurs in the tongue itself, and also being anxious that the patient should be able to retain his speech, I removed only the right half of the tongue and cleared out the enlarged glands in the neck. I desire to draw your attention to the fact that I accurately bisected the tongue from behind forwards before commencing to remove the half of the organ. I lay special stress upon this point because, if you look at the tongue, you may think that I left much more than half of it. The fact is that the tongue has been drawn to the right side. Having removed the half of the tongue, I sewed the cut margin of the lingual mucosa to that passing from the under surface of the tongue to the inner aspect of the lower jaw. For twenty months the patient was absolutely free from any trouble at all, and I was beginning to hope that the disease had been cured, but, unfortunately, there then developed, on the opposite side of the floor of the mouth, a firm induration. This was not glandular, it was clearly an ill-defined infiltration in the floor of the mouth, and for that reason I feel quite certain that we had better leave the case alone; the infiltration was quite without limit, it had passed into the surrounding tissues, and has increased in size rapidly. The patient has, you will see, wonderfully free movement of the tongue. It is frequently stated that if one half of the tongue is removed the rest of the organ becomes bound to the floor of the mouth, but it all depends on the way in which the tongue heals. If, as I did, you stitch the two cut margins of the mucous membrane and obtain primary union the remaining half of the tongue retains free mobility, and speech is, as you hear in this patient's case, not very much affected. Even now, though he has got a large lump in the floor of the mouth, he can talk quite well. One thing I have noticed in this, as in several other cases, namely, that after the removal of one half of the tongue, and when the mucous membrane of the floor of the mouth has been sewn to the dorsal mucosa of the tongue, the tongue becomes too long; thus in this case the patient used at first to bite the tip of his tongue until he learnt to curl it round inside the dental arch, as you see he does. In the present case, then, you see there is recurrence on

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the opposite side of the floor of the mouth, and I have asked myself whether it would not have been better, after all, to have removed the whole of the tongue and to have cleared out both submaxillary triangles. The actual result of the operation, to my mind, is encouraging in that for twenty months the patient has been perfectly free from pain. He has acted all the time as a valet, and has done his work quite satisfactorily, and is even now performing his duties, though this recurrence causes him a certain amount of pain.

Case 6.—A Case of Excision of the Elbow-joint with Good Result.

This next case, that of Ada M—, shows a very encouraging result from very bad tuberculous disease of the elbow. It is always a somewhat doubtful point what to do for tuberculous disease of joints. When I encounter a large tuberculous abscess with unbroken skin I generally open it, and then excise the joint two or three weeks afterwards. When I find septic sinuses with a tuberculous joint I always at first curette the sinuses thoroughly, and paint them with pure carbolic acid to endeavour to render them in that way aseptic. Sometimes I cut the sinus completely out before I start the operation, so as to avoid any septic contamination from that source. This child was admitted into the Victoria Hospital for Children just a year ago, and she had then a very large abscess on the inner aspect of the joint. The elbow-joint was rigid, and owing to pain from ulceration of the cartilages she could not use her arm at all. I made my incision as for an excision of the elbow, opening the abscess as I proceeded. You may think that that is a somewhat unusual incision to make; it is that which I employ in most cases of excision of the elbow-joint, and is the method we owe to Kocher. The incision starts on the outer side two inches above the external condyle, and then crossing obliquely two and a half inches below the olecranon, terminates over the inner aspect of the ulna. It is a curved incision, which gives a far more open exposure than is in any other way obtainable, and with a minimum amount of damage to parts which it is desired to preserve. The abscess contained some twelve ounces of tuberculous pus. We washed out the abscess cavity with sterilised water until it was clean, and then excised the joint. The wound healed by first intention. The little girl has exercised her arm very well, and has, as you see, very nearly full extension, perfect flexion, and a very fair amount of pronation and supination. This result is more than ordinarily satisfactory for a case of excision of the elbow complicated by the presence of a large tuberculous abscess.

Case 7.—Painful Cicatrix on the Back of the Hand.

The next case is one of painful scar. This is a condition which often gives rise to very considerable suffering. You will notice that the patient presents a very large false keloid scar. False keloid scars are, in my experience, very frequently produced by burns from something at very great heat, something much hotter than boiling water. Boiling water will, as a rule, not produce a very firm, dense, false keloid scar such as this. When this man came into my out-patients' room on Tuesday I said at once I thought it was probably done with molten metal or something similar. We found, on inquiry, that it was done two years ago with molten sealing-wax, which attains, I imagine, a very high temperature. The scar remained comparatively painless for about sixteen months, and then became excessively painful, the pain shooting right up to the arm. It is difficult to say why the scar should remain practically painless for over a year and then become the seat of excruciating pain. The explanation that is generally given is that a nerve is involved in the cicatrix. It has always been a difficult point, so far as I am concerned, to understand why the nerve is not so involved in these cases long before it is. In this particular case the cicatrix was probably fully formed in three to four months, and yet it was not until a year later that the radial nerve became painfully involved in the cicatrix. I wish you to notice what one generally finds in these cases, that the involvement of the nerve is only sufficient to strangle it somewhat, not sufficient to paralyse it. The patient has absolutely no anæsthesia at all in the distribution of the radial nerve. Just about this point evidently some small twig of the radial has been compressed, and some neuritis has been set up, as on touching the part there is intense pain shooting up the arm. The treatment in these cases is extremely unsatisfactory. I have in somewhat similar cases dissected out the scar and

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covered the defect by skin grafting, but in almost every case the pain has come back more or less after the operation. The treatment that I have lately adopted in a case of this kind, and which was attended with a gratifying amount of success, consisted in dissecting out and excising two inches of the trunk of the sensory nerve on the proximal side of the scar. A piece of the radial nerve could be easily excised, and this would probably remove all pain, but only at the cost of producing permanent anæsthesia of the dorsal aspects of three and a half digits of this man's right hand. Moreover we should also run the risk of bringing about trophic changes. I only advise this latter method of treatment when the pain is really too severe to be borne. I have found, as a rule, that massage of the scar either very greatly aggravates the pain or very greatly mitigates it. In this case the patient, to whom the *pros* and *cons* have been clearly stated, urgently demands operative treatment, as he says that the pain is truly agonising. It is a case of the choice of two evils.

Case 8.—Tertiary Syphilis.

The last case is one of considerable interest both from a medical and surgical point of view. The patient, R. W—, you see walking, but the characteristics of his gait are somewhat hidden by the fact that he has a perforating ulcer on the sole of one foot. He is suffering markedly from locomotor ataxia. There is no question as regards the diagnosis. He has lost both knee-jerks; he shows, moreover, contraction to accommodation but not to light of both his pupils, and he has lightning pains. When you have a patient with these symptoms the diagnosis of locomotor ataxia is assured. He cannot, as you see, stand with his eyes shut, and he has also zonal pains, and he has had two perforating ulcers. One of them, that over the ball of the big toe, went down to the bone; the other, on the border of the little toe, was not so deep. Though somewhat unusual, one is healing and the other has practically healed. The patient has been resting seven to eight months with great benefit to these ulcers. This case of locomotor ataxia is of syphilitic origin; some books give the percentage of syphilitic cases, but I do not remember ever coming across a case of locomotor ataxia in which one could not be pretty certain that the condition was syphilitic. I do not think there is such a condition as non-syphilitic locomotor ataxia. As a rule the sclerotic symptoms of the central nervous system are the last of all the manifestations in tertiary syphilis. Periosteal manifestations usually occur very much earlier. This patient had his primary sore between twenty and twenty-one years ago. He has, as you will see, very considerable enlargement of the tibia, and he has considerable pain in it, worse when he gets into bed at night, and he has had very frequent periosteal swellings. His periosteal nodes have in part disappeared under iodide of potassium. The patient is taking iodide now in large doses, and as long as he is under iodide the osteitis and periosteitis are naturally very considerably relieved. It is comparatively seldom that one sees a case with anything like the completeness of this one. The patient is certainly better now that he is taking large doses of iodide of potassium. He has been in the hospital three times, and the chief treatment that has been employed has been the administration of iodide of potassium and the removal of the hardened epithelium at the margins of his ulcers by the use of salicylic acid.

WITH DR. E. W. AINLEY WALKER AT GUY'S HOSPITAL.

General Pathology of Fever.—III.

At the end of the last lecture we were speaking of the regulation of the body temperature in its relation to the pyrexia of fever. Such regulation is dependent primarily on the existence of an adequate supply of heat from heat-producing processes. Into these processes we must therefore now examine briefly.

The subject of animal heat was first investigated by Lavoisier, who came to the conclusion that heat-production was carried on by means of the combustion of carbon in the lungs. His view, however, soon became untenable, and was abandoned with the discovery that the oxygen taken up is not combined with carbon in the lungs, but passes on into the blood. This observation led to Liebig's theory that the oxidation processes occur in the

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blood-stream. We cannot enter here in detail into the physiology of the respiratory exchange, but we now know that Liebig also was in error as regards the destination of the oxygen inspired, and that the processes of oxidation, like all other metabolic processes, are carried on both in and by the cells themselves which form the body tissues, and not within the circulating fluids. Hence heat-production is a function of the cellular metabolism. Heat is, in fact, the only constant form in which the energy of the body is expended, and all other forms of energy tend to transform themselves into this form, in terms of which they all may be conveniently expressed.

The great discovery of the principle of the conservation of energy (according to which all energy arises out of previous energy and none is ever lost) by Joule in England, and Meyer in Germany, led the latter sixty years ago to the conclusion that the output of energy by the animal body must correspond exactly to the energy taken in in food. And this conclusion gained wide acceptation, and constantly received support from the experiments of physiologists; but it remained for Rubner to obtain a proof of mathematical precision and completeness. This observer, by a long series of experiments, from which all sources of possible error were carefully eliminated, was able finally to show that the output of energy in the twenty-four hours expressed in calories of heat exactly coincided with the heat value of the food consumed less the heat value of the excrete products.

It has been further proved that the chief source of heat-production is the muscular metabolism. This is established by the following facts:—The ratio of the work done by the whole body to the heat produced is about one to four. The ratio of the work done by a muscle to the heat which it produces is also about one to four. But all the work of the body is done by muscles; hence they produce also practically all the heat. This is the meaning of the normal muscle tonus.

A small proportion of heat is also produced by glandular activity, and, on the authority of Mosso, by the central nervous system.

We now perceive the meaning of the shivering or rigor associated with the onset of acute fevers, and of the increased tone of the muscles at this period even in cases where there is no rigor. It is the immediate cause of increased heat-production, and is associated with the resultant rapid rise of body temperature.

Thermogenesis, therefore, is a function chiefly of the muscular metabolism. What is the relation of the central nervous system to this process, and how is the regulation of the process carried on?

Liebermeister showed that if an individual be placed in a bath at body temperature, so that no heat can be lost except by respiration, yet the body temperature does not rise, at any rate at first. It follows, therefore, that the normal heat-production can be diminished by the nervous regulation.

Then Pflüger showed (about 1880) that if the body temperature be slightly lowered by exposure to cold, there is an increase in the respiratory exchange evidencing an increased heat-production. But if the experiment be done on animals, employing equal artificial respiration first on a normal animal and then after the muscles have been paralysed by an injection of curare, it is found that in the latter case there is a fall of temperature on exposure of the animal to cold, showing that the power to react to such exposure is dependent on the muscular metabolism of the normal animal.

Some ten years later the subject was again investigated by Zuntz, a former pupil of Professor Pflüger. Zuntz showed both on himself and others that the reaction to cold is partly conscious, and is controllable by the will. That is to say, that if a man shivers under the influence of cold his heat-production is thereby increased, and if he shudders the increase is very considerable; but if by an effort of the will he restrains the shivering (and this is possible), there is then no increase of the heat-production under the influence of external cold.

The increased heat-production, therefore, is the result of a subconscious psychical reflex, comparable to the usual reflex which leads to closure of the eyes on the sudden approach of foreign bodies; it is a more or less instinctive reaction, but is in no sense a true reflex process.

Thus, then, we see that heat-production *can* be controlled in both directions by the nervous system, either to lessen or to increase the existing rate of output. And MacAlister came to the conclusion that such a regulation was in normal and continuous action. This supposition, however, as pointed out by Burdon Sanderson, is by no means necessary. In the warm-blooded animals the

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supply of heat is always in excess, whether external conditions be such as tend to lower the body temperature or not. Whatever these may be, the body temperature would tend to rise at any moment if the surface loss should cease. If the available supply of heat were not thus in excess of the requirement, there could not be a regulation at all. You cannot regulate a tap to allow a flow of water of, say, a cubic foot a minute if the supply in the pipe can fall below this quantity. And the experiments of Zuntz showed clearly that even though the tendency to shiver on exposure to cold be fully inhibited by an effort of the will, so that there is no increase in the heat-production, yet the body temperature maintains its normal level owing to the natural excess supply of heat.

It is accordingly not necessary to suppose, as some have done, the existence of a thermogenetic centre in constant action, nor is such constant action *à priori* probable. Heat regulation is a development of evolution in the higher animals. And the results of evolution tend to be such as act to the advantage of the animal concerned. This could not with propriety be said of a regulation which necessitated a change of muscular activity with every change of the external temperature conditions, quite irrespective of the existence or otherwise of a demand for muscular activity in the form of work.

Conditions which call for a direct diminution of the thermogenesis are only of rare occurrence in natural surroundings. And the increase of thermogenesis associated with shivering is, as we have already seen, of psychical origin. It is not essential to the maintenance of the body temperature in ordinary circumstances, and it is due not to a lowering of the body temperature, but to the sensation of cold resulting from a diminution of the surface temperature of the skin. The same is true of shivering and rigors in fever. The old and often repeated statement that the fevered patient shivers although his skin is hot, is absolutely without any vestige of foundation. Exact investigations by the thermo-electric method have proved that in these cases the skin temperature is considerably reduced, and that, during the period of accession, while the body temperature is rising that of the skin is undergoing marked and definite diminution. The rigor, therefore, is the consequence of sensory impulses ascending from the cold skin surface, and is exactly comparable to the shivering in the experiments of Zuntz of which I told you, and which is purely psychical in origin.

The increased heat-production of fever as a whole is therefore not the result of the action of a thermogenetic centre, nor of the paralysis of a thermo-inhibitory mechanism. It must be the direct result of a katalytic action of the febrile agent on the tissues, and of the increased katabolism due to heightened temperature.

Distribution of Heat.—The distribution of the body heat is effected chiefly by the circulation. The temperature of a muscle even outside the body may be raised as much as 0·5° to 0·8° C. (1° to 1·5° F.) by throwing it into a condition of tetanus; accordingly the blood flowing from active muscles is perceptibly warmer than that which reaches them. By the constant circulation of the blood through all the tissues the temperature of the different parts tends to be equalised, and heat produced locally is rapidly distributed throughout the body.

Without a circulation the existence of the larger animals would be quite impossible. The heat produced would then only be carried to the surface slowly by direct conduction, and this would mean that while the surface temperature tended to the level of that of the surrounding objects, the heat as one proceeded inwards would steadily increase until a temperature was reached at which the life of cells would be impossible. A condition comparable to this is sometimes seen in the rapid rise of the internal temperature in fever cases after death, when circulation ceases while the heat-producing processes are still in active operation. The importance of the circulation in maintaining a relatively uniform temperature throughout the body is therefore fundamental. It is sometimes too little recognised.

Now we have seen that in the course of fevers the circulation is considerably slowed. And this must necessarily retard the distribution of heat. Hence there will be a greater difference than normal between the temperature of the internal parts and that of the body surface, the former being raised out of proportion to the surface temperature. But an increased temperature means increased katabolism. Thus it follows that the disturbance of the rate of blood-flow tends of itself to increased heat-production, and to an increase in

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the loss of nitrogen, apart from any special katalytic action of the febrile agent.

The actual amount of increase in the heat-production in the course of fever is a variable quantity. In the experiments of May already mentioned it was found to be about 10 per cent. greater than that produced in inanition. Thus in the experiment already quoted the animal in inanition showed a daily waste of 14 grms. of proteid and 10 grms. of fat; while, in a condition of inanition with fever added, the figures were for proteid 18 grms., and fat 10 grms. as before. Reducing these to their heat values we have as follows:

	Inanition.	Inanition *plus* fever.
Proteid . . .	54·3 calories	69·0 calories
Fat . . .	92·0 ,,	92·0 ,,
Total . . .	146·3 calories	161·0 calories

—giving an increase of about 15 calories on a total of about 146, which is approximately 10 per cent. Nebelthau found an increase never exceeding 10 per cent., and frequently a good deal less than this amount.

Heat-discharge. — The discovery by Claude Bernard in 1852 of the existence of vaso-constrictor nerves, and then by Schiff a little later of the vaso-dilators, evidencing as they did the regulation of the circulation, and especially of the cutaneous circulation by a nervous centre, paved the way for Traube's view of fever as a disorder of the process of heat-discharge, and led to what is spoken of as the *retention* theory. This explained the pyrexia as the outcome of a diminished surface loss of heat,—that is to say, an abnormal retention of heat within the body. The diminished loss was attributed to vaso-motor spasm causing constriction of the cutaneous vessels. This Traube believed continued throughout the fastigium of the fever.

Liebermeister, on the other hand, maintained, as we have seen, that the pyrexia was due to the increase of heat-production by the animal. This position is, however, quite untenable. Violent muscular exertion will give rise to an increase of heat-production as great, and frequently much greater than occurs in fever, even up to as much as double or treble the normal; the effect of feeding on a fasting animal is precisely similar, yet no pyrexia results because the normal regulation leads to a proportionate increase of the heat-discharge.

It is this regulation and the regulating mechanism which are at fault in fever. The existence of a regulation is, as we have seen, only made possible by the fact that the heat-producing processes tend to exceed the requirements of the organism. And hence it is that a disturbance of the regulation leads to pyrexia and not to a diminished temperature.

Only on the failure of the heat-producing activities as death approaches does pyrexia give place to collapse and subnormal temperature where these occur. Conversely, whenever the body temperature rises, regulation is at fault. This is the crucial fact which must be firmly and clearly grasped to obtain a true conception of the pyrexia of fever. It is not that heat-production is increased, though this occurs; nor that the discharge is diminished below the normal, for this is not the case during the fastigium; but that the two opposing mechanisms are no longer balanced, which gives rise to the pyrexia. The regulation is disordered so that the discharge is not co-ordinated to the heat-production; not *enough* heat is given off in fever.

Discharge of heat occurs by conduction, radiation, and evaporation from the surface of the body. A proportion, probably less than one fifth of the whole according to MacAlister, is also lost in the expired air which has been heated in the naso-pharynx. This latter portion is under no direct control; it varies with the temperature of the air breathed—colder air requiring more heat to raise it to the normal temperature—and with the rate and amplitude of respiration. In animals which do not sweat—as, for example, dogs—it is a most important factor in the thermolytic process, but in man it plays a comparatively unimportant part.

The surface loss of heat is governed firstly by the vaso-motor mechanism, since the rate of heat-loss from the surface is dependent on the state of dilatation or constriction of the cutaneous vessels and the rate of flow within them. It is governed also by the so-called sudo-motor or hidrotic nerves, the nerves which regulate the secretory activity of sweat-glands. These nerves, which have been investigated more especially by Langley, were dis-

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covered by Luchsinger. They take their origin from the same region of the central nervous system as the vaso-motor nerves, follow a similar course, and have a very similar distribution. But that they are entirely independent of the latter system is shown by the occurrence at times of sweating associated with constriction of the cutaneous vessels—the so-called cold sweats,—and by the fact that stimulation of certain nerves—for example, of the peripheral end of the divided sciatic of a cat—may produce vaso-constriction and sweating of the pad of the foot at the same time.

In the process of the conversion of water into aqueous vapour a large amount of heat becomes latent or is absorbed, and hence a considerable portion of the surface loss of heat from the body is got rid of in the evaporation of the sudatory secretion. This is the means by which the body temperature maintains its normal level in the presence of great external heat where loss by radiation and conduction have become impossible, as, for example, in the hotter chambers of a Turkish bath. And this explains why great external heat is much more difficult to tolerate in a moist atmosphere where evaporation is limited—as, for example, in certain parts of India—than in a drier climate.

In conditions of health, whenever circumstances tend to cause a rise of body temperature above the normal, these two nerve systems, the vaso-motor and the sudatory nerves, exert a prompt and an efficient regulating action, and by increasing the discharge of heat at the body surface prevent disturbance of the normal temperature. This action fails in fever. In many fevers the skin remains throughout remarkably dry, owing to a cessation or great diminution of its secretory activities. In some, however, as in rheumatic fever, there may be considerable perspiration going on. The feeling of heat imparted to the hand by the skin of fever patients is in great part attributable to its abnormal dryness, which improves conduction, for what is estimated by the hand is not the actual surface temperature, but the conduction rate of the part with which it is in contact.

The cutaneous circulation is disordered in a similar manner. In the accession of fevers which begin with definite chills or rigors there is undoubtedly a spasm of the cutaneous vessels; this is well seen in ague. The rigor, as we have already seen, is the result of sensory impulses of cold ascending from the cold anæmic skin. It may at times be re-induced, especially in the early part of the fastigium, by an exposure of the surface of the body, which leads to a rapid cooling of the skin. The arterial spasm, however, does not persist, as Traube thought, but yields in the fastigium to a condition of variable and varying dilatation of the vessels with diminished rate of blood-flow. With the establishment of defervescence, and especially marked when this occurs by crisis, there appears a re-establishment of sudatory activity with copious perspiration, and a marked dilatation of the cutaneous vessels with a rapidly increasing flow of blood to the surface,—that is to say, a restoration of the thermolytic process to its normal capability.

The characteristic feature of the fastigium is the great variability of the cutaneous vascular conditions, so that even rough measurements show differences in temperature between the different parts of the skin surface at one and the same time, and between the same part tested at different times, such as are quite unknown in normal health.

The paresis of the vaso-motor system which occurs in fever is further evidenced by the fact that the vessels do not react to stimuli so readily as in the normal animal. So long ago as 1870 Heidenhain showed that in fever reflex stimulation of the vaso-motor centre does not, as in health, give rise to an increase in the cutaneous circulation, but rather the reverse. There is no vaso-dilatation. And in continued fevers one can readily demonstrate the fact that the reaction of the vessels is very sluggish by drawing the finger firmly along the skin. The line thus pressed upon becomes pale, and the pallor lasts for several minutes, fading quite gradually and spreading slightly at the edges in the meanwhile.

The condition of the cutaneous vessels in the course of fevers has been investigated accurately by Maragliano with the plethysmograph, and by the thermo-electric method by Geigel. Geigel used a pair of junctions of German silver and iron, one pair being applied to the skin of the patient and the other kept in a vessel of oil at constant temperature. The variations of the skin temperature were indicated by the amount and direction of the current produced. This method gives the actual skin temperature; all thermometric measurements are entirely unreliable for this purpose; they

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are affected by the underlying tissues, and give a record of the temperature an indeterminate distance underneath the skin.

The observations carried out by Geigel proved the following facts:—During a rigor the skin temperature sinks very considerably, so that the difference between the surface and internal temperatures increases rapidly. In the fastigium the surface temperature is only very little greater than in health, but it is much more variable; it is not uniform in the same part at different times, nor identical in different parts at the same time. The commencement of the defervescence is signalised by a rise of the skin temperature due to the now increasing circulation through it.

Similar results were obtained by Maragliano with the plethysmograph applied to limbs. The accession of an attack is marked by definite diminution of the volume of the limb, the result of vascular constriction; while increased volume due to increased circulation precedes the occurrence of the crisis and appears some time before the critical sweating commences. And there may be a gradual and steady increase in volume throughout the latter part of the fastigium. During the fastigium there is on the whole a condition of moderate vaso-dilatation.

Although, as we have seen, the regulation of temperature is disordered in fever, so that not enough heat is lost to balance the increase of heat-production, yet the thermolytic mechanisms are far from being entirely paralysed. The loss of heat is actually increased in fever, and frequently the increase is very great. It may amount to as much as 50 per cent. above the normal rate of heat-discharge according to Richter. This is the meaning of the continuous though variable condition of vaso-dilatation during the fastigium, a condition which contrasts most strikingly with the persistent vaso-constriction seen in cerebral pyrexia, of which I shall speak directly. Were it not so the increased heat-production would of course result in a continued rise of temperature terminated only by the patient's death.

The thermolytic mechanism, therefore, is not paralysed, but only weakened in its action and control. It may be stimulated to renewed activity by certain powerful drugs usually referred to as antipyretics. These were discovered by Filehne some twenty years ago, and he himself investigated the properties of the first two which were obtained—kairin and antipyrin. Their action is, as Burdon Sanderson puts it, translating the expression of Filehne, to open all the sluices for the escape of heat from the surface.

Experimenting with such drugs Geigel has shown that the diminution of the internal temperature which results from their administration is preceded and accompanied by an increase of the skin temperature. And Maragliano found with the plethysmograph that in similar circumstances a dilatation of the peripheral arteries accompanies the fall of temperature; and that as the effect of the dose administered passes off the reappearance of pyrexia is initiated by a diminution in the volume of the limb. Richter, again, using the method of calorimetry, found that the loss of heat increases under the influence of the drugs in question some 25 per cent. above the previous rate of loss in fever, and about 75 per cent. above the normal rate of heat-discharge in health. These three independent observations, therefore, show alike that the action of these drugs is to increase the rate of thermolysis, and this occurs by stimulation of the vaso-dilator centre in the central nervous system.

The experiments also serve to show most clearly, as Burdon Sanderson distinctly pointed out, that the mechanism which regulates the loss of heat at the body surface is capable of giving rise to alterations of the body temperature as great as any which occur in fever. And hence it follows that there is no evidence to be obtained, from this direction at any rate, of the existence of a thermogenetic centre whose function is to exercise a *continuous* control on heat-production, and whose activity is disordered by the action of the febrile agent.

Since pyrexia is not due essentially to a diminution of thermolysis, nor necessarily results from increase of the thermogenesis, but is the outcome of disturbance of the regulating mechanisms which normally adjust and balance these two processes to secure an even normal temperature, it follows that pyrexia will occur not only in fever, but also under every other condition which similarly disturbs the regulating centres. This is well seen in certain cerebral injuries. Its meaning was most thoroughly discussed by Dr. Hale White in his article on "The Theory of a Heat Centre from the Clinical Point of View," in the 'Guy's Hospital Reports' for 1884.

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The influence of the nervous system over body temperature was first investigated by the great physiologist Claude Bernard, who found that after division of the cervical cord an animal becomes incapable of normal maintenance of its body temperature. The observation, however, does not afford any evidence on the question of the existence of so-called calorific nerves, since the result is capable of explanation as due to the complete paralysis of the muscles and of the vaso-constrictor channels of control, leading, on the one hand, to diminished heat-supply from diminution of the muscular metabolism, and, on the other, to an increased loss from the dilated vessels of the skin.

It was, however, observed by Wood, in 1880, that injuries inflicted on the brain in the neighbourhood of the great basal ganglia were followed by considerable rise of temperature. This was confirmed by Aronsohn and Sachs, who showed that the supposedly existing thermic centre was situated in the corpus striatum and the optic thalamus, as had been claimed by Wood. Still later Hale White showed in a whole series of experiments that the centre in question is limited entirely to the former of these ganglia, and lies especially in its anterior end. Lesions confined to the optic thalamus alone did not produce pyrexia at all. The area in question corresponds to the so-called nodus cursorius of Nothnagel. Wood showed by the calorimeter that in these cases there is an increase of the heat-production. And in his Croonian lectures for 1897 Dr. Hale White recorded two clinical cases in which a unilateral paralysis was associated with unilateral pyrexia on the paralysed side, accompanied by unilateral increase of sweat on the same side and increased surface temperature as estimated by the flat coil of a mercurial "skin thermometer;" while there was no visible appreciable difference in the vascular conditions on the two sides of the body.

Such observations have been taken to show that cerebral pyrexia is the result of a disturbance of a nervous centre whose normal function is the regulation of the heat-production. MacAlister in his Goulstonian lectures in 1887 had also summed up in favour of an independent regulation constantly controlling thermogenesis; and incidentally put forward the conclusion that the heat-forming processes in muscle are altogether independent of its motor functions.

The question is a very complicated one, and one which it is hardly possible to answer definitely in the present state of knowledge. But the following considerations are of great importance, in a sense adverse to the conclusions of MacAlister:

Firstly, in cerebral pyrexia there is throughout a marked constriction of the superficial vessels; these are in such conditions so incapable of responding normally to a rise of body temperature that if the rabbit used for the experiments be placed in a hot chamber at 37° C. it rapidly becomes hyperpyrexial, and yet its ears remain quite pale from the persistent vascular constriction present,—that is to say, its surface loss of heat is markedly reduced by vascular spasm. Similarly, by reducing surface loss of heat in healthy animals by keeping them in a chamber at 37° C., their temperature may be brought up to fever heat and there maintained. And hence pyrexia could be accounted for by diminution of the thermolysis. Further, the increased heat-production of cerebral pyrexia is not so great but that it might be explained as the *result* and *not* the *cause* of the raised body temperature, since increase of the temperature alone leads of itself, as we have previously stated, to increased katabolism,—that is, to increase of the heat-production.

Secondly, we have seen that if the thermolytic mechanism is in order it is capable of dealing with very great increases of the thermogenesis, as great or greater than occurs in cerebral pyrexia, without permitting any rise of body temperature.

Thirdly, as Gottlieb found, in cerebral pyrexia the action of such drugs as antipyrin, though it increases the discharge of nitrogen—that is to say, leads to increased katabolism,—yet brings down the temperature to normal by giving rise to an increase of surface loss by means of vascular dilatation precisely as it does in fever cases. From which it follows that even when the heat-production is above the normal, a normal temperature can be maintained so long as the thermolysis is not impaired.

The increased sweating on the hotter side in Hale White's cases by no means proved that there was no diminished loss of heat upon that side, but only that the lesion of the regulating centre was so placed as not to have involved the sudatory mechanism. This being uninjured, an increase of sweat-formation would naturally accompany the

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unilateral pyrexia, which it would not abolish, since sweating alone is insufficient to discharge the amount of heat to be got rid of if a normal temperature is to be maintained. This latter statement is completely proved by the phenomena of rheumatic fever.

The point at issue can only be decided satisfactorily by the use of the plethysmograph upon such cases in order to determine the exact condition of the peripheral circulation, and by the application of the thermo-electric method of determining the surface temperature. The mercurial skin thermometer, as MacAlister has pointed out, does not by any means give a true reading of the surface temperature, but records that of the parts some little distance in below the skin, where obviously the temperature is over normal in such cases.

On the whole question Burdon Sanderson sums up the evidence adversely to the theory that there exists a special thermogenetic centre which is constantly in action. But whatever be the view which we accept upon this question, it is essential to clearly recognise that such pyrexia as occurs in certain cases of head injury is not fever. It has no stages such as characterise a fever, but the temperature rises gradually to reach its height and then gradually again subsides. There is no rigor, and the vascular conditions are not those of fever, in which throughout the course of the fastigium the vessels show a moderate dilatation. It is not preceded nor accompanied by a nutritional disturbance; and, most important of all, its cause is not the same, but is entirely different from the cause of fever.

The question of the etiology of fever is still an open one in its minuter details, but certain valuable evidence is forthcoming on this subject. It had already been found that septic material or the products of acute inflammations were capable, when introduced into the subcutaneous cellular tissue or the circulating blood, of giving rise to fever in the inoculated animal. And with the development of bacteriology and the discovery of the relation of bacteria to infective processes the view arose that the pyretogenetic or fever-causing property of such substances was dependent on the presence in them of bacteria. Burdon Sanderson, however, was able so long ago as 1875 to prepare from putrefying animal matter bacteria-free substances which none the less were capable of giving rise to fever. To the material thus obtained he gave the name of pyrogen. It was prepared by destroying all bacteria and precipitating at the same time most of the proteid matter of the tissues used by means of alcohol; the fluid was then filtered off, and the filtrate evaporated to dryness to remove the alcohol and redissolved in water. Its property of causing fever was removed by filtering it through porcelain. From the results he came to the conclusion that the substance causing fever might be a body analogous to the enzymes. A little later Edelberg discovered that fibrin ferment can similarly give rise to fever on injection; and it was subsequently found that all commercial enzymes will produce the like result.

Kühne, however, was able to determine that the absolutely pure enzymes which he prepared have no such action. This he accordingly concluded must be due to the proteid-disintegration products present in such enzymes as ordinarily prepared. He further showed that albumoses derived from various sources, and notably the deutero-albumose which he obtained from samples of tuberculin, possess a similar action. And Krehl has found a fever-causing albumose in cultures of the *Bacterium coli communis*; while Matthes has obtained a like result with albumoses formed in ordinary digestion. Rouques, moreover, showed that similar bodies can be extracted from fresh animal tissues, and hence that normal cells contain materials capable of giving rise to fever-causing substances. And all the evidence submitted goes to show that these are either albumoses, or at any rate so intimately associated with the albumoses as to be precipitated with them from solution. Their action in the living body is to cause a disorder of metabolism associated with disturbance of nutrition, and to produce derangement of the thermotactic centre in the brain, causing impairment of the thermolytic mechanism.

Fever, then, is not, as the old writers thought, a state of over-action of the normal functions, but is a process characterised, as Virchow claimed, by partial loss of power—an interference with the normal processes.

The pyrexia is not the effect of increased heat-production, as was taught by Liebermeister, although the heat-production is in fact increased; nor is it due to a diminished loss of heat, as Traube held, for the heat-loss is not diminished but is actually increased. It is the result of a disorder of the regulating mechanism by which these processes of thermogenesis and thermolysis are normally adjusted. And this disorder of the thermic regulation is both preceded and accompanied by a disturbance of the nutritive exchanges similar in kind though greater in degree than that which is observed in inanition. Further, although a special heat-production centre may perhaps exist, no evidence in this direction is to be obtained from a consideration of the events of fever nor of the phenomena of pyrexia in general.

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TWO LECTURES ON SYPHILIS.

Delivered at the Polyclinic.

By JONATHAN HUTCHINSON, LL.D., F.R.C.S., F.R.S.

LECTURE I.

GENTLEMEN,—We will begin to-day with a few remarks as to the nature of primary syphilis, and the peculiarity of it. To begin at the beginning, I may refer to the speculations as to the nature of syphilis and its origin, and its introduction into England.

I take it that it is now well established and recognised that syphilis stands side by side with the exanthemata; that it is a febrile disease having quite definite stages—quite as definite as those of smallpox or measles;—and that it must have, necessarily from that fact, a particulate virus, a virus which breeds in the blood, and which produces the various phenomena associated with the disease.

And I think we may infer from that further, for it is quite a legitimate deduction, that this virus has determinate stages of life, that it pursues its life career for a certain period, and that it then dies. That gives the basis for a clear conception as to the nature of syphilis,—that it differs from smallpox and measles and the other exanthemata simply in this, that the duration of its stages is very much longer. There is this difference, that we have no evidence that it can ever be conveyed by aërial infection, that it is contagious only. Smallpox is inoculable in the same way that syphilis is; but smallpox is capable also of being communicated through the air, whereas fortunately syphilis is not.

As to the introduction of syphilis into Europe, my belief is very definite—namely, that syphilis was introduced into Europe at the end of the fifteenth century, with the landing of Columbus. I do not think Columbus brought it for the first time from the West Indies, because the dates do not exactly fit in. In 1493 Columbus returned to Spain, and

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there is no doubt that he had met with a disease of the nature of syphilis in the West Indian Islands where he had made his landing. There is no doubt that his sailors were infected by it, and very probably those sailors had brought it home with them. But it was prevalent in Naples so early as 1494. That is too soon for it to have travelled so far. As we have said, 1493 was the date of his landing, and you will see it is too short an interval for us to imagine that syphilis could have spread from Spain to Italy. I have very little doubt that it was introduced prior to that time from the West Coast of Africa, with which Portuguese traders were in intercourse.

So it was brought from the two places at almost the same date. And we now know that there is at both places an indigenous disease which we believe to be the same, and which, though hitherto known under different names, is identical with syphilis. Then it spread very rapidly. We hear of it in Naples; I do not think it had been previously noticed in Spain, but in Italy it was early recognised. It spread thence in connection with the army of Charles VIII of France, which was then in Italy. When that army returned two years later to Paris it took syphilis back with it. I do not believe anybody reading the facts of history of that time can have any doubt that syphilis was quite new to Europe. Everybody thought it was new when it appeared in Paris, when Charles's soldiers brought it with them two years later, just before the end of the fifteenth century. I repeat, everybody thought it was new. Now I appeal to common sense; is it possible that a disease like syphilis, so conspicuous, so certain to attract attention, so certain to spread, considering the state of morality at that time—and at the present time also for the matter of that, I am afraid,—is it possible that such a disease as syphilis is, if it had existed, could have been overlooked? I say it could not possibly. Everybody thought it was new. And it spread north and west, up into Scandinavia and other European countries, and to England. The date of its appearance at these different places is very fairly well established. I feel certain it was a new disease. We may gain a little corroboration of this from the fact that the poets before that date are silent about it. They were good observers of social habits, very free in their comments upon the customs of their day, and very ready to take note of the inconveniences resulting from lax morality; they were very likely, or I should say certain, to have pointed the finger at such a disease as syphilis had they known of it. Shakespeare, a century later, gave us full descriptions of it; he knew all about it. Chaucer in England, and Boccaccio in Italy, would have referred to it if they had known of it, but they are silent; no one, so far as my knowledge goes, has referred to it prior to the dates I have mentioned. Therefore I assume it was a new disease, introduced at that time. It was at first by no means a purely sexual disease; it was conveyed in various ways—as, indeed, it is at the present time, although much less frequently. At that time the people were careless about it; they did not know its seriousness, and were not careful in their habits. Thus by kissing and various other means syphilis was communicated freely from one to another. It was very frequently communicated to children, much more frequently than it is now, and by non-sexual means. It was then just as contagious as it is now. So at first it was not recognised as having any particular stigma attached to it, though in a very short time it was. I mention those facts because I think they are pretty conclusive in the direction of showing that the disease was a new one in Europe at that time. I may just say here that I believe the disease has not improbably been since very frequently re-introduced from its aboriginal habitats. It is a disease which is endemic on the West Coast of Africa and in the West Indies, and in many other places—as, for instance, in the Fiji Islands. It was not introduced from the Fiji Islands, because we had no intercourse with the inhabitants of them. But in the other places I have mentioned it was endemic among dark races. I think its aboriginal home was in various places. I think very likely it has been re-introduced since that time repeatedly by sailors and others who have visited them. I am taking it for granted here, and I think it is now proved, that the disease which has been called "Yaws" and "Frambesia" at various places, is really none other than frambesial syphilis. With regard to our sailors having contracted "frambesia" in foreign parts during the last two centuries, if there was any new disease there which they could have brought home, I contend that they would have brought it. But they have not done so; they

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have not brought us "yaws," they have not brought us "frambesia;" but they have brought us syphilis, because yaws is the parent of syphilis, and the two diseases are really the same.

We come now to a more obviously practical matter—the consideration of the stages which this syphilis fever observes.

Those stages have been classified, as all present know, into primary, secondary, and tertiary. We shall never set that classification aside, although we may fully recognise that it is not one which can be carried out with very great strictness.

But we will use our words lightly, and will not attempt to be too definite in their limitations.

We know what we mean by the primary stage of syphilis; we mean the stage of inoculation or introduction of the virus characterised usually by the formation of what is known as a primary sore —a chancre. The disease is almost always acquired by inoculation on some part of the skin or mucous membrane; or it may now and then be introduced straight into the blood by subcutaneous injection. The latter must be exceedingly rare, and can occur only under very exceptional conditions. What we know as the primary stage is the formation of a sore which will prove infective, usually associated with irritation in the proximal lymphatic glands.

Next we have what we shall continue to call the secondary stage, which is the febrile stage, the exanthem stage, during which an eruption comes out on the skin of the patient and on the mucous membranes, and during which there is a certain amount of febrile reaction. The intensity of this fever is great in some cases, very slight in the majority, but present more or less, I take it, in almost all. In many cases the amount of fever is quite imperceptible to the patient, but still it is a definite thing.

The secondary stage, then, is characterised by an eruption on the skin and mucous membrane, and by pains in various parts and inflammations in almost all parts—a consequence, we assume, of the poison in the blood. So the primary or first stage is a local one; the second stage is a blood stage, in which it is in every portion of the blood.

And I might stop for a moment here to prove the existence of the virus during the secondary stage in every portion of the patient's fluid tissues, by what has been observed in vaccination syphilis. In vaccination syphilis it has happened that under the hands of a very careful vaccinator, a man who carefully avoided taking blood at all as far as he could observe, keeping himself to clear lymph,—it has happened, I say, that syphilis has been conveyed; and not only to one patient, but to a dozen at a time. The syphilis was of the ordinary kind and of the ordinary severity and course. The realisation of such facts will leave us with a very clear vision that the virus must be existent in all the fluids. If it can exist in such a minute quantity of lymph as is taken in vaccination without drawing blood, then it may be assumed to be present in all the fluids of the body. I do not stay to dispute as to whether or not the white cells of the blood may have been present in the vaccine lymph; I think that probably they were. Many other instances might be adduced in proof that the blood contains the virus, and that the most minute quantities will be sufficient to inoculate the disease when it is in its greatest vigour. This, then, is the secondary stage, during which the virus is present in every portion of the patient's blood, and probably in all his solid tissues also. It breeds very freely, and no portion of the patient's tissues could be taken at that period which would not be found to contain it, and to be capable of conveying it to another person. I have assumed that the virus dies after a while. I think it is a fair assumption that if it has been thus existing through the whole of the patient's body for a certain period, it undergoes death.

The secondary eruption will be accurately symmetrical, proving clearly that it is a blood disease.

When we come to the third stage, that stage which is fortunately most often omitted, but which may occur at any time after the primary and secondary, and to which the name tertiary is given, the symptoms are no longer symmetrical. In this stage they no longer imply blood disease, and the patient is no longer capable of communicating or transmitting the virus to any one else. To this very definite group, containing a great number of dissimilar things, we apply the name "tertiary." It is a disease of the tissues,—that is to say, the symptoms of the tertiary stage concern the solid tissues and not the blood.

We will now proceed to consider the peculiarities which attend the inoculation; in other words, the primary sore.

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We will first ask the question, is it necessary, in order that the virus of syphilis shall gain access to the system, that there should be a breach of the surface? To that I reply in the negative. I think that although a breach of the surface, such as a sore or a wound, or a previous ulcer, is exceedingly favourable to the introduction of the virus, it is by no means essential. I have no doubt that in the large majority of cases—the commonest form of syphilis, that in which it is contracted on the sexual organs—no breach of surface at all occurs.

At any rate it will be within the experience of everyone that those who have contracted syphilis were quite unaware that any breach of surface had occurred. Men who know that they have been in risk, and have examined themselves carefully from day to day for weeks afterwards, tell us that they have never been able to detect any ulcer whatever. In a large majority of cases no ulceration can be detected, and we may assume it is almost certain that the virus may be absorbed without there being any breach of surface. This will depend upon the part to which the virus is applied. The mucous membrane of the sexual organs of both sexes is the part which affords special facilities for such absorption, being thin, delicate, and moist, protected often by folds, so that the virus is not easily wiped away afterwards. It is a part which is abounding in glands, and we can imagine the virus will easily enter into the orifices of glands and be lodged there. At any rate it is a matter of common experience that during the three weeks following exposure no sore is observed. Of that we can be in no doubt whatever. As to the differences seen in the effects of the virus, we may say that these will depend partly upon the region to which the poison is applied, and partly, and very largely, upon the idiosyncrasy of the patient.

We all know that what we call chancres differ very much in their features, and these are the two explanations which I would chiefly offer in reference to it. As to the region—on the genitals in both sexes, but much more definitely in the male sex than in the female,—the delicate mucous membrane, especially the mucous membrane beneath the prepuce and immediately surrounding the corona, is particularly liable to become sclerosed and indurated under the influence of the syphilitic virus.

We might here say that it is a peculiarity of syphilis, throughout the whole of its stages, that it has a remarkable tendency to induce proliferation of cells and the formation of indurations. Sometimes it appears to simulate growth, producing hypertrophy; but it is exceptional that it will induce suppuration. It does not cause suppuration primarily, whilst it is very apt to induce indurations alike in the secondary and tertiary stages. In the primary stage this tendency to produce indurations goes on to the extent of producing definite sclerosis of a most peculiar kind.

To this fact very great importance has been attached. In our book descriptions of chancre it is sometimes implied that the primary sore of syphilis is always indurated and sclerosed. Let me insist that there are very many exceptions to this. We assert only a general principle. Syphilis does tend to produce very remarkable sclerosis in some cases, but it does not do it in nearly all. Nor does it do it in nearly all parts. For practical purposes the most definite examples of sclerosis in connection with the primary sore may be said to be met with only on the genitals. It is said that on the female genitals you scarcely ever meet with indurated sores. That is a mistake. You do very often meet with them. They are frequently overlooked, and they are seldom so characteristic as those which we meet with on the genitals of the male. On the genitals of the male we often meet with a form of induration, especially just above the corona, as hard as cartilage, which is quite specific, and which, to those who have become acquainted with it, is pathognomonic. If we see that, we recognise syphilis. When the prepuce is retracted and you find an indurated chancre, you know that it is an infective one. A few marked exceptions will occur in which even this induration is imitated by lesions which are not primary sores. There is the "recurred chancre," which occurs in persons who have had syphilis years before, and it comes exactly in the site of the original sore. On that very site another may form without any fresh inoculation whatever. It looks exactly like a primary chancre, and no examination enables you to discriminate between them. You have to go into the history of the case and form your diagnosis. In proof of the statement that the part of the mucous membrame involved has much to say to the occurrence or otherwise of what we call specific

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induration, we may mention that the application of caustics to this part will sometimes induce an induration which might easily mislead. It never, however, produces induration of the cartilaginous type, but there will be a good degree of hardness, which might very easily mislead. The application of caustics, such as nitric acid to warts, will produce a condition apparently like an infective chancre, and it is therefore very necessary to beware of the fallacy.

We have now to come to the great question of the diagnosis between true infective chancres and chancres which will not affect the system. Our forefathers were very much less careful in this matter than we are now. They assumed that all sores on the genitals were syphilis, and they commenced the use of mercury very early indeed,—that is to say, as soon as they saw the sore on the genitals, those who believed in mercury commenced a mercurial course. And in that blundering way I think it possible that they sometimes did more good than we do now, simply because they began their treatment more early. Of course by such a promiscuous procedure many patients got a course of mercury who did not deserve it or need it; but, on the other hand, the really syphilitic patients got the specific very promptly. However, we have got beyond that now. Fifty years or so ago the distinction between the hard and the soft chancre was strongly drawn—that is to say, the distinction between what I prefer to call the infective and the non-infective chancre,—and surgeons were accustomed to talk of the "*ulcus molle*." It was thought there was a dualism in syphilis—that there was one kind which was manifested by a "soft sore" and another by the hard sore. I think we may now say—as, indeed, I said a great many years ago, much to the evident disapprobation of some of my specialist friends—that the doctrine of dualism in syphilis is dead. I think it is now really dead, for we do not nowadays hear anybody talking about dualism in syphilis. We recognise that there are sores which are non-infective as well as those which are infective; but that there are two kinds of syphilis, and that the word "dualism" is in any way applicable, no one nowadays dreams of. It would be quite as logical to say that there are two kinds of vaccine virus, because in some cases the vaccine sore inflames very much and does not develop in its typical way. The difference is a purely local one. The "*ulcus molle*," if it be truly non-infective, is not followed by anything in the nature of syphilis; it is an abortive inoculation resulting from some venereal discharge, in all probability related to the syphilitic one, but not containing the specific poison of syphilis.

We now have to come to the consideration of the stages. And first let me remind you that in investigating syphilis we are not dealing with the introduction of a pure virus. There is no such thing as a pure culture in the case of syphilis; it is introduced in a mixed state in the great majority of instances, and it is owing to that admixture of pus and other secretions in the accidental way in which syphilis is conveyed that we owe the fact that such great multiplicity of appearances result in the primary sores. They are not the results of the introduction of the pure virus. So in order to study the natural development of syphilis I am accustomed to go back to certain cases in which it was probably a tolerably pure virus which was introduced, as in the case of vaccination syphilis. In these cases no products of inflammation were conveyed with it, but simply the poison. Thus we are able in these cases to study the ordinary appearances which a sore assumes when the virus is tolerably pure. If the virus with which the inoculation is made be pure, in a general way no result whatever will follow until three weeks or a month, or even five weeks have passed. I believe that this can be laid down with great certainty. It is borne out also by experiments, some intentional, and many which were almost equivalent to experiments, in vaccination cases. So that we may say that the incubation stage, prior to the appearance of any sore whatever, before the place which has been inoculated will inflame in any degree whatever, will be at least three weeks, very probably a month, and possibly five weeks. For convenience of memory we will therefore say a month, and at the same time allow a little margin on either side, but very little on the side of being shorter.

With regard to the first appearance of the sore, it will be simply a little papule or little red patch or spot, which may be observed for a few days. It will take a week before anything in the nature of specific induration which you can estimate with the finger will be produced,—that is to say, a week or ten days from the commencement of the

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disease. If no sore has appeared until the end of nearly four weeks, it is probable that it will indurate within a week of its beginning to such a degree that it may be recognised as an infective chancre. But as we have seen, it is not always that the virus is introduced in a pure and uncontaminated state. In some, and a great number of cases, some other sores due to the introduction of the products of inflammation, pus and other poisons, are present; and the elements which the pus may contain may produce a sore which will take precedence of the true chancre. Mixed chancres are very common. You may have a patient with a non-indurating chancre which was never at any stage "hard." The absence of induration is all that we should take cognizance of. "Softness" is not in itself a positive feature of the sore—it is only a negative thing,—nor anything pathognomonic to which we can trust. We will suggest that the inoculation has been effected with pus which contained the syphilitic virus. In these the sore may inflame immediately after the application of the poison, and so a sore will be noticed within a few days of the intercourse from which the disease has been derived; and if the patient is then seen by an incautious practitioner he may perhaps be assured that he has not got syphilis—that he has not got the specific disease—because there is no induration. But it is possible that this "soft chancre" may heal by the application of a little iodoform, and then at the end of a month induration may take place. That is a course which is quite possible, and which is, indeed, very frequent. In the cases of vaccination chancres, which give us excellent illustrations of the uncomplicated malady, the virus was introduced simultaneously with the vaccine virus. The course was this: the vaccine virus took. There was the ovoid vaccination sore well characterised, with beautiful vesicles, running its course in the orthodox way, ending by scabbing over, the scab drying and falling off, and the vaccinator believing that everything was going on as well as possible. Then, after it had soundly healed, at the end of a month from the date of vaccination the patient began to experience a little itching, and began to rub the place because it was irritable. Then a little red pimple formed, and in a week or two there were produced sores such as those of which the illustrations are before you. These illustrations show vaccination chancres in different patients, but at almost the same stages, and you will notice the characters produced. The course was the same in them all, or nearly so. In a few cases the vaccination places had remained inflamed during the whole period—that is to say, through the whole month,—scabbed over and inflamed and unable to heal; but in the larger majority—in all the patients in one batch—it had quite healed, and the vaccination was believed to be most satisfactory, until at the end of the month the sore indurated and reopened, and syphilis followed. What do we prove by this? We prove that the syphilitic virus is not prevented from exerting its full efficiency or power by existing in company with some other poison; that that other poison may produce its stages of inflammation and go through its periods before the syphilitic virus comes into play. So, arguing from what we observe here, we may infer that it is quite possible, when a pus inoculation has induced a pus sore, that that also may go through its stages and may end, and the specific virus may exert its force at the end of its proper period. And that, I believe, is very commonly the case. At any rate, for practical purposes I warn everyone most strongly never to tell the patient who has got a venereal sore, or one which may possibly have been contracted by a venereal source, that you are sure he has not got syphilis, until a month has elapsed, for it is not till then that you can judge. The syphilis may be absolutely latent until a month, or it may be entirely concealed by the presence of something else, such as an inflamed sore, until that period has elapsed. Hence my idea of the relationship between the non-indurated sore and the indurated—that the sores which are not indurated are the result of various forms of pus contagion, and that they may very often carry with them also the virus of syphilis.

It is necessary now for me to say a few words as to the peculiarities which are presented by the sores called soft. They vary very much indeed. Multiplicity is one of the features in which the non-indurated sore differs from the indurated. The secretion of pus is another. These are features which it is well to keep in mind. But an indurated sore may also be multiple, and four or five up to fifteen indurated sores have been noticed together. And on the other hand, of course, non-indurating sores may be single. But in proportion to the shortness of the period intervening between the

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contagion and the appearance of the sore may you feel sure that that sore is not as yet syphilitic. This does not exclude syphilis afterwards, but it makes it pretty clear that that sore as at present seen is not due to the syphilitic virus; that virus produces nothing until at least three weeks have elapsed.

Next with regard to the occurrence of glandular swellings in the groin. In the true indurated Hunterian chancre it is usual for the glands in the groin to be enlarged and to become very hard. The expression "bullet bubo" is a very proper one. The chancre may be as hard as cartilage, and the swelling in the glands of the groin may be as hard as bullets, quite movable,—that is, not becoming glued together. Here, again, the tendency of the syphilitic virus is to cause an adhesive inflammation with fibrinous exudation, a tendency to organisation and not to suppuration; whereas in the other form there is a tendency to suppuration. But just as the sore itself is inflamed and ulcerated, so the bubo which results from it is inflamed and tends to suppurate. But none of these features of distinction must be pushed too far. The bubo of true syphilis may also suppurate, and this we witness not very infrequently. There is a chance also, of course, that the other form may not suppurate; indeed, it is only exceptional that it does so.

I do not think any very useful purpose would be served by attempting to describe the varieties of non-indurating sore. Frequently they have abruptly cut edges, and I have mentioned the feature of multiplicity which often attends absence of induration. They vary very much in different cases. They are not as a rule under the influence of mercury, unless they be complicated by the syphilitic virus. They are as a rule under the influence of local treatment, and remedies of which iodoform stands at the head.

To continue our description of true syphilis, we have to consider now in what directions of deviation from the type of the indurated sore the true infecting sore may vary. It may vary within very wide limits, and we have to ask the question, is it possible for syphilis to be induced without any sore at all—what is called in France *syphilis d'emblee?* And I should be obliged to answer that in this way—that so far as one's facts go we must admit that syphilis may be originated without any chancre. Certainly it is not very frequently originated without any chancre which either the patient or the surgeon could ever appreciate, even by the most careful examination, and this in patients who were perfectly frank. It may be that sometimes the sore is within the urethra; in some cases there is some suspicion with regard to this, but it is very seldom that anything is to be found in the urethra which would lead you to locate it. There may be a little discharge, but it is very infrequent that there is any large amount of evidence to that effect in the urethra. We have all seen cases in which, as I have said, although most careful observation has been made, no chancre whatever has been recognised.

A useful point in diagnosis is to examine the glands. If you find hard glands in the groin, the inference is there has been a chancre at the anus, on the perineum, or on the genitals; and if you find a hard bubo in the armpit, that the hand has been the seat of infection. Whilst I believe that there is such a thing as syphilis without any obvious chancre, I have no doubt there is in most of these cases some trivial sore, and it must in some be trivial to a minute degree, because observant patients and surgeons often fail to identify it. In the cases of surgeons who get syphilis after having their hands exposed in midwifery practice, and who yet never recognise a chancre, I suppose the virus has probably been lodged by the side of the nail; I can vouch for a considerable number of such cases.

I alluded in the beginning of this lecture to the possibility of syphilis being caused by subcutaneous injection.

I have known cases in which surgeons have become the subjects of syphilis from rather deep pricks with needles which were poisoned. In one case I saw the patient during the whole course of his disease. He had pricked himself in the thumb with a needle when operating on a syphilitic patient. He was very anxious about it. The site of the prick was a little tender for some time, and then there was just the slightest brown discoloration around it. That was all he had in the way of chancre; he never had any sore, and never developed any induration, only a little discoloration. My reason for asserting my belief that sometimes the syphilitic virus may be absorbed without producing a local sore is to introduce the

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statement that in a great many cases the sore is not a characteristic one. We are far too much in the habit of insisting that the primary sore of syphilis should always be a well-characterised indurated chancre. In very many cases it is not so. And here, again, it is necessary to be cautious in pronouncing that any sore which is venereal is not syphilis, or may not become so. If we can have syphilis without a chancre in the case of pricks, it is possible it may follow sores on the genitals which never indurate, and I am sure that it frequently does so. In the diagnosis of primary syphilis we of course pay great attention to induration, for when it is present it is a symptom which is beyond appeal. In a great number of cases we can be certain, but in a large number we must remain more or less in doubt, and we must give our opinions with caution. If there be no induration whatever in the glands, that is another reason for doubting specific inoculation. If the sore disappears very quickly, and there is throughout no induration of glands, that is a stronger reason; but the omission of induration of glands is not infrequent in syphilis, and we must not, therefore, trust too much to it.

I will next speak as to the duration of induration, and as to its influence by remedies. Nothing is more positive than that mercury will cause the induration of a primary chancre to disappear. You may experiment upon that. Any one who has any doubt as to the influence of mercury upon syphilis, or upon the sclerosis produced by it, may easily convince himself in these cases. Give a patient with an indurated sore mercury, and it softens within a few days and then melts away; stop your mercury, and it indurates again. Resume your mercury, and it softens. There can be no doubt about that matter. As to the question whether a chancre will disappear without treatment, unquestionably in the large majority of cases a chancre will disappear without any treatment whatever. These are the cases in which the patients will tell us that they have had no syphilis, and that they have never been aware of anything in the nature of a chancre which they could recognise as such. In their case the primary chancre has disappeared spontaneously. I believe that a primary chancre will usually disappear spontaneously in a month or six weeks. But it is difficult in the present day to get facts which help us in this direction, because everybody gives mercury as soon as the disease is recognised, and all the cases which come under our observation are complicated by the drug. I have but few data upon which to base the estimation of how long the period of induration would last if there were no treatment. But the induration would certainly go away, except in the very rarest cases, after a period of a few months; at the utmost we may allow six months. The secondary stage of syphilis will often begin while the primary one is still existent. It is the rule, if the patient be not treated, for the symptoms of blood poisoning to begin to manifest themselves long before the primary induration has disappeared. A very peculiar feature indeed is the recrudescence of indurated chancres. I have said that if you stop mercury, after having pushed it so far as to induce softening and almost complete disappearance of the sore, it will re-indurate. But what I meant by "recrudescence of the chancre" is its reappearance spontaneously, after a considerable period, in the precise locality in which it first appeared, without any fresh inoculation whatever. In these cases the induration induced is exactly like that of the original one. So any one who is not familiar with the fact that a recrudescence is possible would certainly be inclined to doubt his patient's statement, and to believe that he had contracted a fresh sore. But this phenomenon is most undoubted. I believe I was the first to notice it. But almost at the same time, within a year of the publication of my first notes on the matter, the same thing was described by Professor Fournier, of Paris; and we shall have much to say about this in speaking of tertiary symptoms and discussing whether the chancres recurring in the site of the original ones shall be termed gummata. They appear to be a perfectly local phenomenon.

I think it will be well for us to next advert to two or three other peculiarities which the primary sore may assume. One is that it may cause not a hard ulcer, but a fungating growth. Here is such a growth depicted, surrounded by a ring of induration; and in one of my vaccination cases it was still more evident. Those who have seen the last number of our 'Polyclinic Journal' will perhaps have noticed a report of a lecture of mine in which I mentioned that the chancres which result from inoculation of syphilis in the act of tattooing very

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frequently show a mass of granulation tissue like a little raspberry. This is very important, because it has been this kind of sore that has been claimed as peculiar in the case of yaws, which disease I feel sure is nothing but syphilis. It is therefore interesting to note that the primary sore on the skin in English syphilis is very often frambœsiform.

Another point is this: occasionally a sore will heal. Whilst one may say that a primary chancre may sometimes never ulcerate at all, we must admit that sometimes it is nothing but an ulceration. Primary chancres have been cut out in mistake for tumours because they were not ulcerating at all, the skin around remaining perfectly sound and soft. I have said that the most specific collar of induration is most frequently seen on the male genitals, and that induration is very frequently omitted in other places.

Now we come to speak of erratic chancres, those which are contracted on some other part than the genitals, and usually quite accidentally, and we have to ask what are the peculiarities of the chancres under such conditions. It has been customary to say that you never get the soft chancre anywhere but on the genitals, and I find in my own little book on syphilis that I have said that in terms which may lead to some mistake. It is quite true we never identify a sore on any other part of the body as being venereal but not destined to lead to syphilitic symptoms,—that is to say, we do not identify any sores which are not really infective. As a matter of fact almost all sores which occur on the tongue, lips, skin, or on other parts than the genitals, are infective. But these are not all indurated, by a very long way. Very many of them show no induration, and only a few of them are definitely indurated. I have seen on the fingers very definite induration sometimes; but in the majority of cases you must not expect to recognise any specific induration in an erratic chancre. Sometimes they are deeply ulcerated, sometimes fungating, sometimes simply an ulcer. But their conditions are very various, and they are not infrequently attended by suppurating glands. In the operation of circumcision in Jewish families syphilis is sometimes communicated by careless manipulators. I had an opportunity of seeing a whole series of children who had been infected by the same operator In that case the history was that as a rule the wound had healed for a short time—perhaps the healing was a little slow, but it healed in most of them,—and that when it was healed it broke out again, and a large suppurating sore formed, sometimes a phagedenic sore with enlargement of glands in the groin, with excessive suppuration of those glands. In none was there specific induration.

I have next to mention the possibility of the occurrence of phagedena in connection with primary syphilis. We have said that sclerosis is the rule, and that it is the peculiarity of it. But that is not to be taken to exclude the possibility of ulceration and suppuration. Still further we go, and say that it does not exclude the occurrence of phagedena. Phagedena and sloughing occur not infrequently in connection with the syphilitic virus, and in all stages of the disease. It may affect the primary sore. One hint may here be given as to the possible reason why the sores which do not indurate have been supposed not to induce syphilis. It may be that the mere fact of ulceration of the sore soon after the introduction of the virus removes the virus. If the inflammation occurs within a day or two of the contagion, it may be equivalent to cauterisation. So the phagedena and the sloughing away of the tissue may be equivalent to the excision of the chancre. I am afraid, however, that the occurrence of phagedena in a chancre does not usually have that result. In a considerable series of cases which I have collected in which the chancre became phagedenic, in almost all of them secondary symptoms supervened. The explanation is that the phagedena usually occurs too late; it does not come about early enough to wholly remove the virus by destroying the portion of tissue which has been contaminated.

Then as regards the secondary symptoms and the period of the appearance. We have said, as an aid to the memory, that one month from the introduction of the virus is the date at which the induration will appear. In round figures, again as an aid to the memory, we will say that two months must elapse before the secondary symptoms begin. These dates are, indeed, very nearly accurate. Sometimes they may be a little less, but seldom much less. I speak from a whole series of cases inoculated at the same time—vaccination cases, which serve me with my best facts, and a number

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of others in which I was quite certain of the dates, and in which, therefore, we could strike averages without any risk of error. From these cases I am able to say that about two months is the period at which you must expect the secondary symptoms. The patient will at that time begin to be a little feverish, and will probably have an eruption on the skin, and that eruption will probably be in the first instance roseolous, one very much like that of measles, an eruption which may very probably be overlooked by the patient. That eruption is quite transitory, and it is usually followed, in the course of two or three weeks, by a papular or mixed one. Sometimes the roseolous one may persist more or less whilst the papular one is developing.

I will devote the rest of my remarks to treatment. First with regard to treatment of the primary stage, and as to when that treatment should be commenced. I entertain no doubt, and I believe very few nowadays do, that mercury may be regarded as a specific against the syphilitic virus. In using the word specific, I mean that it has a specific antagonism to the life of the virus which causes syphilis, that it either kills that virus or very greatly restrains its power. We know that mercury is antagonistic to low forms of vegetable life in almost all conditions. The agriculturist uses it to prevent the growth of various parasitic cryptogams; and so in syphilis it is antagonistic to the life of the intruding organism. But very often, in ordinary practice, it does not actually kill it; it restrains its growth, restrains its development, and keeps it back. It invariably manifests its power and its tendency to restrain. It removes the indurated chancre and prevents the secondary symptoms. Of the latter fact I make no question whatever, because I have been for the last fifteen or twenty years in the habit of giving mercury very early, and giving it with the intention of entirely anticipating and preventing the secondary phenomena. In a very great number of cases, a very large majority, it is effectual for that purpose. The symptom which it least constantly prevents is the sore throat; and, indeed, mercury often causes sore throat, and you may be a little confused on that point. If a person comes under care with an indurated sore and mercury be given, and given quickly, it makes the sore disappear; and if the mercury is steadily pushed to the full extent that the patient will bear it, and continued, you may be tolerably confident that no secondary symptoms will occur. I will except sore throat, but there will be no skin phenomena whatever. The temperature, if it has been high, will subside at once, and the discomfort and bone-aches will disappear. Thus I make no question whatever that it is possible to suppress syphilis by an early treatment with mercury; nor have I any doubt that it is wise to begin mercury as soon as ever the diagnosis of syphilis is confident. As soon as you feel sure you have to deal with syphilis, begin the use of mercury, and push it to the full extent which the patient will bear. The inconveniences attending the use of mercury are the salivation and the occurrence of diarrhœa; otherwise I think that mercury seldom does anybody any harm. It is true that it reduces the fat a little and makes the patient feel weak, but I have practically never known a case in which the person was really the worse for the mercury given. If you give your patient directions as to diet while he is taking the mercury, he will not have diarrhœa. I forbid most emphatically every kind of fruit, all green vegetables, and tell the patient to live simply upon beef, mutton, fish, and potatoes and bread, and give him an opium pill to take, if need be, to prevent diarrhœa. It is not desirable that the patient should be salivated, because when that happens you cannot push the mercury further. My object is to get as much mercury into the patient as possible, as much as ever the patient will bear, so as the more effectually to check his syphilis. I also insist strongly on long continuance of the course without any intermissions.

Influenza in Children.—The conclusions of W. C. Williams are as follows: (1) Children of any age, even in early infancy, are susceptible to the influenza infection. (2) Repeated relapses and grave sequelæ are frequent in cases in which the initial seizure is mild as well as when it is severe. Therefore, no attack should ever be considered as "mild" nor as recovered from until a sufficient time has elapsed. (3) Clinical diagnosis should always be supported by the evidence of the microscope, whenever possible, as the practical certainty thus obtained may be of great value in explaining future developments.—*Journal of the American Medical Association*, July 4th, 1903.

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LECTURE

ON

METHODS OF PREPARATION FOR OPERATIONS.

Delivered at the Medical Graduates' College and Polyclinic, June 30th, 1903.

By F. C. WALLIS, F.R.C.S.,
Surgeon to Charing Cross Hospital.

THE object of this lecture is to suggest methods for the possibility of making the preparation and conduction of operations a matter of quite simple routine which may be carried out effectively, without regard to the surroundings, and with the certainty of a good result.

The suggestions I propose to make are the results of my own practical experience, carried out over a number of years, after trying most of the somewhat complicated and often drastic measures which have been advocated for the purpose of obtaining aseptic healing.

There is no intention to decry the original methods, which at the time were most useful; the simple details which I propose to submit to you are rather the outcome of these early proceedings. Indeed, this lecture is intended as a plea for simplicity in surgical operations, having regard to the many and complicated procedures which are practised and advocated to-day. In complicated systems the very complications themselves are apt to, and do, defeat the end aimed at; and the more complicated the system the more frequent is the possibility of failure. The points I propose to consider are:

1. The general condition of the patient.
2. The general preparation of the patient.
3. The preparation of the patient's skin.
4. The preparation of the hands of operator and assistants.
5. The instruments.
6. The towels, sponges, and ligatures.
7. The technique of the operation.
8. The dressings.
9. The operating room.

The General Condition of the Patient.—There are three points to which I wish to allude under this heading:—(*a*) The condition of the kidneys and viscera generally; (*b*) the condition of the patient's mouth and teeth; (*c*) the previous surroundings of the patient.

The investigation of the condition of the viscera, especially the kidneys, is supposed to be a matter of routine, but is too often forgotten. A small amount of albumen does not negative an operation, provided the percentage of urea secreted is 1 per cent. or more, and also provided that the operation is not one which may open up large areas of tissue to the possibilities of infection—as occurs, for instance, in Kraske's operation for rectal carcinoma. But when albumen is found in the urine the operation should not be undertaken until a thorough investigation of this secretion has been made, when the question will be settled by what is found.

The condition of the patient's mouth and teeth, especially in hospital patients, is often a matter of vital importance. The teeth are frequently foul, and the gums ulcerated. This condition has probably existed for some time, and the patient has absorbed, swallowed, and inhaled a great deal of septic matter.

People in such a condition are very likely to suppurate, but the great danger here is that already there may be some latent septic condition in the lungs, which the anæsthetic starts into activity, and septic pneumonia is the result.

The previous surroundings of a patient, especially when the temperament is a nervous one, should be considered. People coming from a quiet country place, coming probably for the first time to a great and noisy city, a hospital, and all the, to them, horrors of a surgical ward, should, when possible, have time to accustom themselves to the new surroundings before any operation is performed.

The general preparation of a patient may be, usually is, quite a simple affair, but this depends upon the conditions already mentioned; it is always desirable, when circumstances permit, and especially before any serious operation, to have a patient under observation for two or three days, during which time the diet is restricted, the bowels regulated, and the patient is generally overhauled. Moreover the surgeon and the patient get to know each other—a consideration well worth thinking about.

The Preparation of the Patient's Skin.—The desirability of endeavouring to procure sterile skin, with the methods at present used, is an open question.

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In some lectures by Dr. F. W. Andrewes he there puts in a scientific manner the same ideas which I have derived from my practical experience.

To obtain sterile skin an extremely vigorous process has to be carried out, the details of which are well known to all of us; and with regard to this it must be remembered that all skins are not alike, and what one epidermis will stand with equanimity will cause positive havoc in another.

The usual practice of soaking the skin for several hours with some strong chemical solution by means of a compress, after it has been vigorously scrubbed, etc., often results in the production of a crop of pustules over the operation area, which should effectively prevent any operation being done at that time.

The reason of this is fairly obvious, but I do not propose to do more than state the fact here.

Sterile skin is quite difficult to obtain, and the very means, as at present known, which are used to get this desired condition, produce often exactly the opposite result.

As a matter of fact sterile skin is not necessary, however desirable, for the practical result aimed at by the surgeon. If it were so the percentage of our "aseptic results" would be poor indeed.

In proof of this, let us think for a moment of the hundreds and hundreds of emergency cases of strangulated hernia, intestinal obstruction, and various other acute cases in which an operation has to be performed *at once*. Very rarely, in fact hardly ever, is there any other result than an aseptic union of the wound. This is a matter which I have discussed with a great many surgeons, and their experience is the same as my own. Whereas, in cases in which an elaborate and irritating skin toilet has been carried out, suppuration of a mild, but none the less annoying type occurs, sometimes even eight days after the operation, with a normal temperature possibly. This delays union, makes the result of doubtful benefit, and, in fact, is a failure from the point of view of the surgery of to-day.

The method about to be described has been adopted in all my operations for the last five years, and during that time outside the hospitals I have only had one case suppurate and one stitch abscess. The case which suppurated was one of the early ones, and, although due to carelessness on my part, it was not caused by any skin condition. The cases in hospital that have suppurated have not, except in a very few, been due to skin infection, and, as altogether the number of cases amounts now to many hundreds, the method may be said to have had a full trial.

Since I have given up using strong chemical irritants I have not had any cases of that latent suppuration which I have already spoken of, and this is especially the case in operations for radical cure of hernia—an operation which is a most severe test for methods of surgical cleanliness.

Three hours before operation the skin is washed with soft soap and water, and any necessary shaving is carried out; a boracic compress is applied and bandaged on. At the time of operation the sodden epidermis is removed by means of a rubber brush, the surface washed with ether, and then with lysoform. In emergency operations a stiff brush, used with discretion, is quite as effective as the boracic compress for getting rid of the superficial layers of epidermis.

The Preparation of the Hands.—The surgeon who uses strong solutions for his hands, of either carbolic or some preparation of mercury often combined with spirit, soon has a shiny, harsh epidermis, a mixture of blood and carbolic or mercury in and around his nails, which makes his hands unpleasant to look upon, unpleasant to touch, and they are so rough that the difficulty of keeping them ordinarily clean is much increased. The more a man operates under these conditions the worse the hands become.

If it is necessary that an operation on healthy tissues has to be done immediately after a septic case, then strong measures must be taken, or gloves worn at either one operation or the other; but this is an uncommon chance.

The wearing of gloves at operations is at present rather a fashion, but not likely, I believe, to be a permanent one. It is quite a good plan for the assistant to wear them, but they interfere greatly with the manual dexterity of the operator—so much so that this is a bar to their use, except on special occasions.

Soft soap, hot water, and a sterilised nail-brush are the main things for procuring surgically clean hands; after these have been well applied the hands are further washed in lysol, or lysoform for choice, and this again is washed off with sterilised water; the nails are kept short. It will be noticed

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that the mechanical measures are those mostly relied on in preparing the skin. The above method is to be absolutely relied on, and the frequent operator, if he adopts it, will find his hands at the end of a hard week's work none the worse for wear, and of the same appearance and feel as those of any other man in his station of life.

The Instruments.—These should be as few as possible, and yet sufficient for the operation.

If matters have been thought out beforehand there should be no occasion to find that some instrument essential to the operation has been forgotten. Yet this is what is constantly occurring in hospitals, the reason being that the surgeon does not make himself personally responsible for overseeing what instruments are being prepared for the work in hand; and if this becomes a habit some essentials are apt to be forgotten when operating away from the hospital. The best instruments are the surgeon's hands, when it is known how to use them.

All instruments should have metal handles; this enables them to be boiled, which is the most effective method of sterilising. Some washing soda should be added to the water; otherwise the instruments which are nickel-plated become discoloured. The knives, if boiled at all, should be done separately.

After the instruments have been placed in the steriliser they should not be touched by any one except the surgeon or his assistant. After boiling they are tipped out into the tray, from which they are taken by the operator as wanted. The tray is filled with either carbolic lotion or sterilised water; and personally I use sterilised water only.

When an instrument is not being used it should be returned to the tray; it is a bad practice to have a lot of instruments scattered over the operation area.

Cold water and a scrubbing-brush are the best means of cleansing the instruments and hands of blood; the former are then boiled again for about fifteen minutes, dried, and put away in a clean metal or wooden receptacle, which should never be used for anything else but *clean* instruments. By paying attention to this a very few minutes' boiling at the time of the operation is all that is necessary to ensure sterility of the instruments.

The Towels, Sponges, and Ligatures.—The towels should be dry sterilised ones, where possible. When this is not possible they should be boiled, and then placed in previously boiled water, wrung out, and spread round the field of operation.

Marine sponges are now almost a curiosity at surgical operations. Gamgee tissue, ordinary cotton wool, Turkey towelling, or some specially prepared arrangement of gauze and wool, generically known as "dabs," are mostly used; and these are boiled previously to the operation and placed in sterilised water, from which they are taken by the surgeon. When finished with they are put in a bowl placed conveniently for the purpose, and not strewn about over the towels or thrown on the floor. This is an untidy habit.

Silk is the most reliable of all materials for ligatures; it is strong, easily made surgically safe, and ties the best knot. It can be either boiled and then kept in a carbolic lotion 1 in 20, or else put straight into a 1 in 500 ethereal solution of perchloride of mercury. This latter method is one which I have used now for many years with the best results. It renders the silk absolutely safe, and previous boiling, which makes the silk brittle, is not necessary. For skin sutures silkworm gut is the best material. But for a buried subcuticular stitch either fine silver wire or silk is used.

The Technique of the Operation.—If the previous details are properly carried out, then during the operation there is a surgical cordon drawn around the patient, the instruments, etc., the operator, and his assistant. No one but these two (the operator and assistant) touches anything which has to do with the operation, and these two touch *nothing else.* If this simple plan is carried out, nothing can go wrong. That great care is wanted will be only too well known to the surgeon, who has not only to look after himself but everyone else.

The first stumblingblock is the apron. This may be a sterilised cotton smock or a rubber apron, but whether sterilised or not it must be considered as "beyond the pale" and outside the operation altogether; it is simply worn as a protection to the clothes and for no other purpose; it has, then, nothing to do with the operation, and there are many ways by which it can if sterilised be fouled. Blood is sponged up at once, and not allowed to soak into the tissues. All bleeding points are immediately clipped, not necessarily ligatured; in fact, this is rarely necessary.

The end of a ligature or suture should never be

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lost sight of; otherwise it may trail against the side of the table, the operator's apron, etc. This is a most important point.

The less that healthy cut tissues are soaked in chemical lotions the better. Strong lotions during the operation are not wanted, and are injurious to the tissues themselves, lessening their natural protective power and causing an excessive exudation of serum, which by tension separates the surfaces and prevents or delays healing, and also is an excellent medium for stray micro-organisms to develop in. Healthy tissues do not require any chemical solution applied to them, and unless some septic material is being dealt with nothing but sterilised water should be used, its only benefit being to moisten whatever material is being used for sponging.

The time taken over an operation is a matter of great importance; unless a habit is made of not wasting time, a great deal of it is soon frittered away by speculation as to what or what not to do, discussing, arguing—in fact, doing anything but the work in hand.

This question of time becomes one of considerable importance in operations which must last for about an hour, and which have often to be carried out in people already debilitated. After the first hour, every fifteen minutes becomes of progressive importance with regard to the recovery of a patient, especially in operations involving much loss of blood, when the patient is apt to become extremely collapsed, and the finishing of the operation may have to be undertaken under most disadvantageous circumstances. Haste without hurry should be the motto of the operator.

Much time may be lost by indifferent anæsthetisation. A good anæsthetist makes all the difference to the comfort of operating, the length of time it takes, and the condition of the patient afterwards. It is not in my province to discuss this question of anæsthetics, but nevertheless it is of great importance to the operator. What can be worse than to have to perform an abdominal operation upon a patient who is breathing stertorously, whose muscles are hard and contracted, and who, owing to extreme venous congestion, bleeds freely from every cut capillary? What more annoying than that a patient should begin to retch violently just when it is necessary to keep the intestines in the abdomen? It would take too long to dwell any further on this matter, although from a surgeon's point of view there is a great deal to be said with regard to the help or disadvantage which may be obtained from a good or indifferent anæsthetist, during or after an operation.

The surroundings during an operation should be kept as clean as possible. A number of soiled sponges or dabs thrown on the floor and strewn over the seat of operation, mixed with a dozen instruments, and blood everywhere, is the usual sight at an operation. Each sponge as it is finished with should be placed in a receptacle for the purpose, and as few instruments as possible should be placed on the towels.

All these details are simply a question of method.

The question as to what antiseptic lotion is best is one which is daily discussed; the tide of favour sets towards carbolic one day, perchloride the next week, combinations of biniodide the week after, and between whiles some new germicide is investigated and eulogised. When one thinks of the various compounds that are used before and during an operation, this matter, although easily managed in a hospital, becomes serious in a cottage or small house. To enumerate a few: turpentine, ether, and carbolic, or some mercurial solution for the skin; spirit and biniodide for the hands; carbolic for the instruments, and something else for the sponges. Surely if some one fluid could answer all these purposes it would be a great boon.

Lately I have used with much satisfaction a preparation of formic aldehyde and soap known as lysoform, which appears to possess all the requisite characters of an effective germicide, and possessing few of their drawbacks. It has been vouched for scientifically by various English and German experts, and from my own practical experience I can speak in its favour. The advantages are that the one lotion can be used for everything; the hands are in no way roughened or made harsh or discoloured by its constant use. The objection to it is that, unless the lotion is washed off with water before operating, the instruments are difficult to hold because of the soapy character of the fluid.

The vessels which require a ligature are usually few in number; torsion is quite sufficient for the vessels cut by the incision.

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When tissues have been widely separated during the operation, it is as well to as far as possible obliterate the cavity formed by means of buried sutures, and if these are of fine silk a tiny fragment is all that is left in the tissues.

Dressings should be as simple as possible. Dry sterilised plain gauze is all that is necessary to apply as a dressing to the clean wound, with simple sterilised wool and a bandage. Many surgeons use a covering of collodion for wounds, and this answers very well in most cases; but I do not advocate its use, because of the non-absorbable character of the covering. The great point is to keep the wound as dry as possible; any serous exudation is readily absorbed by gauze, but accumulates often to the detriment of the wound when collodion is used. If sterilised gauze cannot be procured, dry cyanide gauze should be used; and if the wound is in the groin or axilla, or made on a hairy subject, dry sterilised iodoform powder is dusted over the incision. The unpleasant smell of this powder may be disguised with vanilla.

The Operating Room.—The stereotyped preparation of a room for a surgical operation is certainly desirable, but not essential.

Many serious operations have daily to be performed under circumstances which do not permit of an elaborate preparation, and this should in no way debar the operation from having the best results. I have operated in small, dirty, ill-kept rooms for such operations as scirrhous mammæ, ovarian cysts, removal of the appendix, etc., and the results have all been excellent.

If a thorough cleansing of the room can be made in plenty of time—all hangings taken down, the carpet taken up, the floor scrubbed, the walls washed or wiped down, and all unnecessary furniture removed,—these are safeguards which it is as well should be taken. But unless this can be thoroughly done the room should be left alone, as any brushing or sweeping only stirs up the dust and does no good.

It may be possible to still further simplify these details in the future.

X Ray in Cancer.—Allen, writing in the 'Boston Medical and Surgical Journal,' June 25th, says a combination of treatment by cutting and by X-ray reduction is the best method.

AN ASPECT OF THE MORPHIA HABIT IN AN EARLY STAGE.

By A. H. PRICHARD, M.R.C.S., L.R.C.P.

In virtue of the great prevalence of the morphia habit amongst different classes of people, one is struck by the very general treatment which the subject receives, and the lack of detail, especially in regard to early stages of the habit, found in many of the excellent books which deal with opium and its alkaloids. Erroneous ideas are, I think, apt to result from such a general treatment of the subject.

In view of this I have ventured to bring forward a case—an early one it is true, but a case which possesses certain peculiar features,—and it is, I think, of some interest.

During a year I had opportunities of observing a man who was addicted to the morphia habit. For three months morphine had been regularly prescribed for him during the course of a painful and exhausting illness, but on recovering from the malady he unfortunately retained a strong desire for the effect of the drug, which had been of very material use in the treatment of his disease.

At the termination of his illness half a grain of morphine sulphate daily had become necessary, and during the succeeding nine months he had been obliged to double this dose to obtain desired results.

In spite of the speedy toleration usual in the use of opium and its alkaloids, he had not exceeded that amount which after six months came to be his daily dose—one grain. The effect then became to all intents and purposes constant, though necessarily a little weakened towards the end.

It was his custom to administer the daily dose each evening after a light dinner; then followed a period, amounting at least to five hours, during which he was able to engage with success in active mental work, his faculties being rendered especially acute; and ideas which would not ordinarily have been developed, became, in such circumstances, the rule.

His vital centres were obviously influenced, as was evidenced by slower rhythm of the heart, and at the outset by some increase of force of the ventricles, and there seemed to be marked improve-

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ment in regard to control of the vaso-motor system; the man was extremely prone to functional relaxation of the peripheral vessels.

At 1 a.m., as a rule, he retired to rest; during this time the effects of stimulation were naturally obliterated, and his sleep was ordinarily of a heavy kind. Dreams of rather a vivid sort usually occurred, and these at first were merely pictures of ordinary happenings, but later in the year they began to assume a grotesque and an unnatural form; on one occasion he narrated how a hitherto undescribed monster, goaded by some unfriendly acquaintance, had watched him for what seemed an interminable age, and finally, when at the acme of despair, he had been awakened.

At 8 a.m. he arose, and before 9 a.m. was engaged in professional work requiring considerable mental effort.

The effects of fairly marked cerebral stimulation still persisted, but in the afternoon, after luncheon, and about eighteen hours after the administration of the drug, he usually experienced some somnolence, which, however, could be readily overcome did the necessity for physical exertion arise; but if encouraged, sleep of a moderate kind, lasting from an hour to an hour and a half, occurred. This, when terminated, left him in a state of obvious reaction: the pupils were widely dilated, the conjunctivæ dull and suffused; cardiac acceleration, with appreciable weakening of contraction, occurred, and depression of other vital functions was apparent.

Moderate impairment of sensation, though not enough to obviate pain, existed while full influence of the morphia was maintained.

During this period, amounting to a year, the man was suffering from syphilis, which was contracted six months before the necessity for morphia had arisen. At the outset the malady promised to be of a fairly virulent type, but while administering morphia no indications of active syphilis, other than slight secondary affections of the tongue, largely due to the irritation brought about by smoking, occurred. Mercury, however, was administered throughout. No very appreciable impairment of general health or marked derangement of bodily functions was noted; the appetite, though somewhat lessened, remained good, and at times, when the drug had been administered before dinner, it was very distinctly augmented.

Constipation, never a very troublesome symptom, necessarily occurred, but late in the year tremor of the hands, apparent only when the man was not fully influenced, some slight sallow appearance of the skin, and impairment of memory existed.

At the end of a year the man was induced to forsake this mode of existence. Few drugs, by way of relief, were employed: a judicious amount of wine helped in regard to the depression, alkalies with small doses of nux vomica relieved to some extent an irritable and sensitive gastric mucous membrane, and cascara with belladonna, after a week or ten days, dispelled in a great measure the constipation.

Within six days the more important troubles had subsided; marked vaso-motor paralysis, producing a sensation of much cold, lack of appetite, and an intensely irritable feeling at the epigastrium had in a great measure passed off, and within nine days he confidently affirmed that the merely physical cravings had been removed. Three weeks later he appeared to have no desire for the use of morphia.

In the study of this case the following points claim attention:

1. That in the nine months following an illness during which morphine was taken to relieve pain, the dose was only doubled, and that for several months the effect of one grain of morphia sulphate seemed almost constant. Moreover there were no signs finally of any very appreciable weakening of effect, except in the matter of the depressant and hypnotic action, which was, indeed, reduced to a minimum.

2. That very little alteration of the general health was apparent, even after a year's use, and that the course of early syphilis did not seem to be influenced adversely by the drug.

3. That urgent symptoms, after withdrawal of morphine, only persisted for about five days.

The Latest Operation for Incurable Vesico-vaginal Fistula. — Rose gives as his most recent method a procedure which involves (1) the longitudinal obliteration of the vulva, and (2) the production of the so-called "large intestinal urethra," which means that the bladder is drained into the large intestine and the urine evacuated *per rectum*. By this method permanent results are obtained, and the sufferers are transformed from chronic invalids into persons capable of pursuing their ordinary avocations.—*Medical Record*, July 11th, 1903.

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CLINICAL LECTURE

ON

TREATMENT OF PELVIC CELLULITIS.

Delivered at the Medical Graduates' College and Polyclinic, October 13th, 1902,

By G. E. HERMAN, M.B., F.R.C.P., F.R.C.S.

THE treatment of pelvic cellulitis may be divided into four points. First, rest; secondly, nursing; thirdly, medical treatment, the least important; fourthly, surgical treatment. The most important is the nursing. The common history of these cases is that the woman was confined and seemed to be going on well. At the usual time she got up, but found herself so unwell that she had to go to bed again. It is no use telling such a patient to go to bed. She will say, "That is exactly what I have been doing." She has kept her bed, and she has not improved. But take the patient into a hospital where she is well nursed, and she soon gets well. Good nursing means in these cases really securing rest. It is not enough to get a poor woman to lie in bed at her home, because there she does not get rest. Every time she has to reach out of bed to get something, to adjust the bedclothes, or to obtain food—this means disturbance to the part; and therefore these patients do not get well, or improve very slowly, at home. But if the patient has a good nurse, who foresees and supplies all her needs, then she recovers. The nurse does not need any brilliant flashes of insight, but only to have been well trained in her routine duties. If a nurse, well trained by a good institution, is secured, almost every necessity is met.

But notwithstanding good nursing, these patients have symptoms of which they complain; and they expect that medicine should do something for them. What they most complain of is continuous

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burning pain in the region affected; and they want the pain removed. One drug, opium, beyond all question relieves pain, but it confines the bowels, takes away appetite, and interferes with digestion. If you give opium, give it in the form of chlorodyne. Another remedy is local depletion. If you put some leeches over the swelling, and some blood is extracted, relief will often follow. But this treatment is not always desirable, because these patients often cannot tolerate loss of blood. Though leeches may relieve pain, you must look at the condition of the patient before prescribing them. Another remedy highly spoken of, although I myself have used it little, is the application of ice. It seems to me that a bag of ice over the affected part is rather a nuisance unless it greatly relieves the pain, and I have not found it do this. External applications for relieving pain should be such as can be painted or lightly rubbed on, because the patients are too tender to allow of prolonged friction. A good ointment is one made of two ounces of Unguentum Iodi and one drachm of Extract of Belladonna. Some think that the iodine favours absorption, and the belladonna acts as a sedative. Another useful external application consists of equal parts of the Liniments of Aconite, Opium, and Belladonna, Menthol and Chloroform. Remedies have been supposed to produce absorption in inflammation. My own belief is that the effects of drugs in producing absorption of the inflammatory products in pelvic cellulitis are so slight as to be practically non-existent; I think not any one of them shortens the course of the disease. Compresses have been recommended, and there is more to be said for them. One very distinguished man—Mr. Hutchinson—believes in salt-water compresses for alleviating pain in some joint diseases. This remedy is useful in pelvic cellulitis; it can do no harm, and it makes the patient more comfortable. Hot douches are supposed to procure absorption. A hot douche to the vagina may act as a sort of mild counter-irritant, and it does not annoy the patient. When the disease has reached a chronic stage, massage is sometimes recommended. In the acute stage the patient is too tender, but when the disease has become chronic massage may stretch the fibrous tissue, make the parts less rigid, and thus do some good. It must not be practised until the inflammation has subsided. These are the chief remedies by which we can relieve the patient's local pain.

The other points in the medical treatment consist in promoting digestion and helping the patient to take food by giving the ordinary drugs for that purpose. The bowels are generally confined, and therefore it may be necessary—perhaps not every day, because the patient does not take much food—to see that she is not constipated. The food that the patient ought to take will be indicated by her appetite. A little alcohol will help appetite and promote sleep. These are the chief points in the medical treatment of pelvic cellulitis. When you get the patients well nursed, support nutrition and relieve pain,—in more than half the cases within a month the exudation will be absorbed, and the patient will be convalescent.

But not all cases end in a favourable way. Some suppurate, and when an abscess forms it must be opened. The swelling may persist long without any sign of pointing. The proper surgical treatment is to let out the pus with the least possible delay, because the patients will remain ill until the pus is liberated. They are generally well in about a month after the opening of the abscess. Among some cases that I tabulated from the records of the London Hospital, the suppurated cases were but little longer in recovering than the cases which did not suppurate. The reason was because the abscess was opened within a few days of the patient's admission.

The difficulty in some of these cases is that you may have a swelling in the lower abdomen, which there is reason to suppose contains pus, and yet there may be uncertainty as to where the pus is.

There has been conflict of opinion with regard to this disease, because until about 1860, or even later, almost every inflammation in the pelvis was called "pelvic cellulitis." The distinction between cellulitis and pelvic peritonitis was not known. About that time Bernutz and Goupil showed that nearly all the pelvic inflammations which up to that time had been called pelvic cellulitis were really in the peritoneum. Then Mr. Lawson Tait showed that many such cases were due to suppurated Fallopian tubes and ovaries, and could be cured by opening the peritoneal cavity. This caused a reaction of opinion, and made some think that there was no such thing as pelvic cellulitis. They opened the abdomen in cases where

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a pelvic swelling was present, and found to their astonishment that the peritoneum was perfectly healthy, and that the disease was outside it.

An abdominal swelling due to suppuration in the cellular tissue is usually on one side. It feels as if close under the abdominal wall, the long axis of the swelling being parallel to Poupart's ligament. A swelling produced by a suppurated tube or ovary, which is large enough to be felt by the abdomen, will be almost central in position, and does not feel as if it were continuous with the pelvic bones. A swelling due to pelvic cellulitis, when felt bimanually, feels like a band of induration continuous with the uterus at one end and the pelvic bones at the other.

When these physical signs lead you to believe that such a swelling is in the cellular tissue, the way to open it is by an incision parallel to Poupart's ligament. When Mr. Lawson Tait first discovered the frequency of tubal disease, in his enthusiasm for abdominal section he advocated opening the abdomen and draining any abscess from the middle line of the abdomen. But that introduces an element of danger by exposing the peritoneum to contact with pus.

In some cases the seat of suppuration is the cellular tissue between the uterus and bladder. In that case the swelling between the uterus and bladder is easily made out. If there is a distinct swelling between the uterus and bladder it should be opened up by an incision through the anterior vaginal wall. Cut through the mucous membrane with scissors, then separate the bladder from the uterus with the fingers, and in doing so you will open up the suppurating cavity. Such cases are rare. I have seen a few cases in which there was an irregular temperature, pelvic pain, wasting, and deficient mobility of the uterus, without any definitely bounded swelling either in the inguinal region or at either side of the uterus. In such cases I have found benefit follow making an incision through the anterior vaginal wall, and then freely opening up the broad ligament with the fingers.

There are rare cases in which suppuration takes place low down by the side of the uterus. Most cases of this kind of cellulitis arise from non-puerperal cases, chiefly operations on the uterus. They are rare, and it is to be hoped that they will get rarer as each generation passes. In these cases there are no physical signs on abdominal examination, except tenderness and rigidity of the muscles. On vaginal examination a swelling is felt on either or both sides which joins the uterus low down at the level of the insertion of the vagina, and slopes off downwards and outwards to the pelvic wall. The swelling produced by pelvic peritonitis joins the uterus at the level of the internal os, and at the sides as it is followed up it feels as if it retreated out of reach. Felt by the rectum a swelling due to pelvic peritonitis is convex, but the swelling of cellulitis surrounds the rectum like a half ring, which feels as if it were continuous with and fixed to the pelvic bones. When this kind of cellulitis suppurates, its usual place for pointing is through the sacro-sciatic notch, but it may burst into the rectum or into the vagina. When the uterus is fixed by inflammatory exudation low down, and an abscess is discharging into the rectum, vagina, or externally, which does not seem inclined to heal, extirpation of the uterus is good treatment, because in this operation the broad ligaments are freely opened up, and so the suppurating cavities can be drained and allowed to heal up. But such cases are rare. Most pelvic abscesses can be drained without removing the uterus. I have seen a case treated successfully by removing the uterus, but I have never had occasion to do it in such a case myself.

Professor Freund has described a form of chronic parametritis which he thinks is from the beginning a chronic disease, and never acute. I have never seen such a case. Cases of chronic cellulitis that I have seen have been the remains of acute attacks.

Pelvic cellulitis is sometimes secondary to disease of the bladder or bowel. In these cases the cellulitis is altogether a secondary matter to the disease of the bladder or bowel which produced it. Therefore I have nothing to say of such pelvic cellulitis.

I have referred to massage as a possible remedy when pelvic cellulitis has passed into a chronic stage and does not get well. I have seen abroad what they call uterine massage. The operator sits by the side of the patient, puts his finger into the vagina, presses up the uterus, and kneads the abdomen for some ten minutes. I have never seen any satisfactory evidence that this does good. If beneficial it ought to be carried out by a nurse.

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Pelvic Peritonitis.

Other kinds of pelvic inflammation result from micro-organisms getting access to the genital mucous membrane. The commonest causes of inflammation of the genital mucous membrane are septic organisms and the gonococcus. Some such cases have been said to be the result of the pneumococcus and some of actinomycosis. There are cases of tubercular disease of the Fallopian tube. There are cases of peritonitis which occur in virgins in whom is no reason to suppose that they have been infected by the gonococcus or by septic organisms. These cases, I suppose, are the result of some micro-organism, but we know not what it is. There are two ways in which these micro-organisms can infect the patient. They may at once directly attack the tissues, or they may find in the genital passage a suitable food which enables the organisms to multiply and acquire such virulence that they can then attack the tissues. This results from the retention of secundines, lochia, or sloughing tissues in the genital canal. About these effects of micro-organisms there cannot be the slightest doubt. Where patients are kept under strict aseptic conditions these pelvic inflammations are never produced. We have no complete knowledge of the micro-organisms which produce septic diseases. Even if in the discharge you identify the streptococcus you have learnt next to nothing. I have had under my care three cases, in each of which there was a large pyosalpinx. One of them had some pneumonia. It was a grave question whether the patient should have an operation done or should wait for the pneumonia to get well. We waited a few days and the pneumonia got rather worse. We thought it better to deal with the abdominal condition; the abdomen was opened, and we found a large pyosalpinx. It was ruptured in removal. The pus was examined bacteriologically, and was found to contain living streptococci, staphylococci, and bacteria coli. The patient had her peritoneal cavity deluged with these living organisms; but she recovered without a bad symptom. I have had two other cases in which living streptococci were present, and poured into the peritoneum in abundance without harm. On the other hand, I operated on a case in which the patient had been ill only a few days. The symptoms were so acute that a diagnosis was made of tubal pregnancy. The tube contained pus, which was examined and found to be sterile. Such astonishing contrasts are so great as to prevent one from being guided by the presence or absence of any particular bacterium. It has been proposed by a Vienna professor that on opening the abdomen and finding a collection of pus the surgeon should first aspirate the pus and have a bacteriologist on the spot to examine it and ascertain what organisms are present, and so guide the surgeon in his treatment. But that is going far ahead of our present knowledge. The cases I have related show that the discovery of the streptococcus would not be of clinical value.

The gonococcus is an interesting study. Sir Thomas Barlow pointed out in his address at Manchester how the clinical history of diphtheria was made clear by the history of the bacteria which caused it. Many irregularities in the effects of gonorrhœa have now been made clear by investigation of the habits of the gonococcus. The gonococcus is rather an aristocratic parasite, because it only affects man. No attempt has yet been successful to inoculate one of the lower animals with the gonococcus. It is an organism which requires a highly organised culture material: it disdains simple nutriment. It grows best at the temperature of the human body. It is killed by twenty-four hours' desiccation. That is a most important and valuable thing, because were it not so the gonococcus would be diffused far more widely than it is. It can live for hours in sterilised water, and several minutes in ordinary solution of chloride of zinc. The gonococcus flourishes most in healthy mucous membrane. We see the most terrific consequences where the gonococcus has infected the conjunctiva of a newly born child. The first attack of gonorrhœa, for the same reason, is more severe than a subsequent one. The gonococcus can only affect parts where the epithelium covering is thin; it does not produce any effect where the epithelium is thick, or there is epidermis. That is why the gonococcus is so often accidentally inoculated in female children but not in males. The mucous membrane of the vulva is thin, whereas that of the male organ is thicker, covered with the foreskin, and thus protected from the organism. The most frequent sites at which the gonococcus gains access are the urethra and the cervix uteri. Gonorrhœal pus has been placed in the vagina without producing any effect. So

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far as we know there is no such thing as immunity to the effects of the gonococcus. Gonorrhœa often gets well without treatment; the gonococci lose their virulence and die, and all the effects of the disease may pass off. But when the gonococcus has in successive generations in one soil lost its virulence and is then transplanted to another, it gains further strength, and may become as virulent as ever. That is why a man who believes himself cured of gonorrhœa may nevertheless impart it to his wife. Although free from symptoms he had some gonococci remaining in his urethra. Or a man who has never had gonorrhœa may get it from a woman who has been apparently cured. The only way to ascertain whether the organism is present or not in the urethra is to examine the secretion of the mucous membrane and make cultures from it. One negative result is not enough to prove the absence of the gonococcus.

The wide-reaching effects of gonorrhœa were first brought to the attention of the profession by Nœggerath, of New York. Until his time gonorrhœa was regarded as practically a disease of prostitutes and bachelors, but we know now that among females who suffer from the effects of gonorrhœa the prostitutes are but a fraction. Nœggerath deserves credit, because he called attention to the fact that gonorrhœa is a more serious thing than it was then thought to be. Nœggerath stated that only one case in ten was really cured. That is an exaggeration. The experience of general practitioners in this matter, if we could get it, would be of extreme value. Specialists only see the sequels; they know not how many cases there may be without any sequels. The same remark applies to syphilis. If some family doctors would each publish notes of every case of gonorrhœa or syphilis treated by him, and the results to wife and children of subsequent marriage, we should be able to get a more accurate view than we now have of the consequences of these diseases. I think there is no general practitioner of experience who cannot recall numbers of men who have been under his care for gonorrhœa before marriage, and yet their wives have remained perfectly healthy and been fertile.

The part of the female genital tract first affected depends on the local condition. If the vaginal orifice is small the probability is that the gonococcus will get first access to the urethra; if it is large the cervix uteri may be the first part to suffer. When gonococci get access to either the urethra or the cervix uteri they generally, but not invariably, attack the vagina. The disease is a transient condition in the vagina, although it lingers long in the urethra and cervix uteri. Therefore a woman may have gonorrhœa in her cervix uteri but not in her vagina, and a man may have connection with her and yet take no disease, while another man may have connection with the same woman and get infected. In the adult infection by the gonococcus is usually direct, but in female children indirect infection is commoner, the child being inoculated by a dirty towel, or dirty fingers, or in some other way arising out of want of cleanliness. One can imagine that the gonococcus may in the past have been communicated by either the surgeon's fingers or instruments. Some have therefore said that the surgeon should not use a glass speculum, because the edges of it might get rough and retain the gonococcus. I think that is seldom the case, because desiccation soon kills the gonococcus. Gonorrhœa of the mouth in infants has been described, but I have never seen a case. Infection of the rectum is said by those who have looked for it to be common in women, because the infective material may trickle from the vagina to the anus. I have not seen such a case. In children, when the gonococcus is inoculated it begins to multiply within twenty-four hours, and the disease is well established within forty-eight hours. In adults the time between inoculation and the appearance of the symptoms of the disease is much longer. One explanation is that only a few organisms may be inoculated, and that they produce not much effect until they have had time to multiply. Another more probably correct explanation is that the organisms may be of little virulence, and until they have multiplied and acquired virulence from a fresh soil they do not produce marked symptoms. This explains why the onset of the disease may be delayed for several days. When the gonococcus has invaded the epithelium it penetrates between the epithelial cells in clumps or lines. When it has got below the surface the leucocytes come out and attack the gonococci; there is a battle between them, and the result is the shedding of the epithelium. If the gonococci have got deeply into the diseased tissue they may get into the blood-stream and produce those arthritic affections associated with gonorrhœa. The processes following gonococcal infection, whether in the urethra, the vagina, the cervix uteri, or the Fallopian tube, are identical.

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TWO LECTURES ON SYPHILIS.

Delivered at the Polyclinic.

By JONATHAN HUTCHINSON, LL.D., F.R.C.S., F.R.S.

Lecture II.

Gentlemen,—I will begin to-day by showing you photographs from the museum. The first shows a chancre of the upper lip and the eruption coming out coincidently with the chancre, or rather before the chancre had disappeared. We said in the last lecture that the primary sore might vary very much in character. I have seen a primary chancre which involved one half of a man's face, and the whole of it disappeared under syphilitic treatment. In the next photograph you see how large a primary sore may be, and how different from the ordinary conception of a primary chancre. Here is a deep ulcer with a sloughing base almost, and very widely extended. There was no question about that being a primary chancre. There was a swelling under the jaw, a bubo; and secondary symptoms followed it. It occurred in a very respectable young woman, who in some way was accidentally infected on the cheek. In the next photograph I show you a very extraordinary case, almost the only case that I have known in my own practice of a patient in whom we could not cure syphilis. We gave this poor man mercury, we salivated him repeatedly, we gave him iodides, and resorted to all kinds of treatment, and sent him to the seaside for months to undergo the treatment there; but all the mercury he had by inunction and by the mouth did him no good. We did not try the injection plan, but we brought about the full influence of mercury into his system. Sometimes he seemed to get somewhat better, and at other times he was worse. The iodides all seemed to make it much worse. I had him under treatment for a year or more—I should think for nearly two years from the beginning of his syphilis,—but we failed to cure him. I do not know what became of him in the end. He was a very respectable man, and it was quite a casual occurrence that he had contracted syphilis. In the photograph you see the bossy mass which the disease presented. It was a secondary eruption of bossy growths, which I suspect were increased by the iodides. I often thought, indeed, that it was an iodide eruption to some extent; but it was originally syphilitic. It occurred on the skin only; and I might remark in reference to syphilis that in the secondary stage, and sometimes throughout the whole of the disease, the stress of the malady seems to fall on one particular set of tissues—sometimes on the mucous membranes, such as in the nose and mouth,—the patient remaining well with regard to the nervous system and bones. Sometimes the stress of the disease falls upon the skin. In the case illustrated the man had no other manifestation of syphilis except recurrent ulcerations with hard bossy edges and hard masses. We tried local treatment with iodoform and all such applications, we cauterised him over and over again with nitric acid, all of which treatments he bore very patiently; but at the end of my acquaintance with him we had failed to cure him. It is one of the cases which specifics do not agree with, fortunately a very small class indeed. In my experience I have met with exceedingly few patients with whom mercury did not agree and in whom one could not produce the wished-for result with the use of mercury and iodide. I never use iodides in the secondary stage unless there is something very peculiar in the condition. If there were bone mischief I would use iodides; but I should avoid them in the secondary stage and trust to mercury unless the conditions were very exceptional.

I show you next a portrait of what is sometimes called syphilitic rupia. A rupial eruption used to be accounted a tertiary eruption because it was attended by ulceration, but we now make no distinction as regards the character of the disease; whether it is merely exudative or congestive, or whether it is ulcerative, we do not say that that entitles us to classify one as being secondary and the other as tertiary. We know that ulcerations occur in some persons in the early stage of syphilis, and everything that occurs in the early stage of syphilis, during the time that the syphilis is a blood disease, is of course classed as a secondary symptom. We distinguish between the stages more by time than by the phenomena which result: the primary during the first two months, or as long as the chancre may last; the secondary during the whole of the time that the patient's blood is infected, and that the eruption is coming out as a symmetrical eruption, as a general eruption due to

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blood disease. I show you a good portrait illustrating the condition known as rupia, which we now, I think, recognise as a form of secondary eruption and not tertiary, occurring usually within four to six or eight months of the primary disease. The earliest eruption is usually erythematous, papular, and superficial; and then later on, in those who are predisposed to it, ulcerative (of which rupia is the chief), in which a vegetation forms, followed by heaping up of crusts. These crusts may be compared to a limpet shell, one with an elevated point. It assumes its form in a way which can be easily understood if you imagine that the little ulcer which is under the crust is gradually spreading at its edges, has lifted up the apex of the crust, produced the next layer, and then spread still further and lifted up the original apex of the crust. Rupia usually leaves scars, not very conspicuous ones, which we know as shilling scars because they are often of the size of a shilling and about as round—nummular scars which are very characteristic of a rupial eruption. It is curable by the use of mercury, but not by iodides, although I will not deny that if the case resisted mercury one might try iodide in connection with it. As a rule, however, the worse the form of syphilis the more definite is the indication for the free use of the specific, and that specific is mercury.

Next I show you a drawing of what I shall have to allude to presently—namely, the occurrence of pemphigus in connection with syphilis, which is a very rare event indeed. There is a bullous eruption which you could not distinguish in the least from that of pemphigus. The condition which precedes rupia is a little vesication; it is not a big bulla containing serum, and these do not form rupial crusts. Of course it might run into rupia, but as a rule it remains pemphigus.

My last picture belongs to a very peculiar case which I shall have to describe, in which a man in perfect health who contracted syphilis was treated for it in the usual way. He got almost rid of his eruption, and so he went on for years in good health. He was a married man, and had no severe symptoms of any other kind; there was merely this persisting slight skin eruption. Many people told him it was not syphilitic, but there was no question that he had had syphilis; and finally it passed very accurately into the condition shown here. He came to me in this state, and I said, "It must be syphilitic still." This was eight or ten years after his syphilis, during which he had been liable to slight eruptions on the skin, which had increased during the last few months. I showed the patient to my son, and he said, "Of course it is syphilitic; there can be no doubt about that." We treated him with mercury. I had tried mercury and iodides, but could do nothing for it. Finally, to shorten the story, I asked my friends Dr. Radcliffe Crocker and Dr. Liveing to meet me in consultation and say whether it was syphilis or mycosis fungoides. The case had resisted inunction. We had taken him into a home and had given mercurial baths, and tried hot-water baths and inunction at the same time, fully salivating him; but we never did him any good. Dr. Liveing and Dr. Radcliffe Crocker met me in consultation, and we had a most minute discussion as to the case, and it ended in one of them saying he was sure it was syphilis, and the other that it was mycosis fungoides. My opinion is this—and you will notice I take a middle course,—that it was a syphilitic form of mycosis fungoides.

We now go on with our consideration of the secondary symptoms of syphilis. We mentioned in last week's lecture that the primary sore was very often not a characteristic one, that it was a great mistake to suppose it was always an indurated Hunterian chancre, and that it might vary very much. We suggested that the variations in the condition of the sore depended not in the least upon any difference in the virus which produced it, but on the patient who received it. Everything depended upon the patient, upon his idiosyncrasies and proclivities. And that statement, which is, I think, the clue, and a valuable one, to the interpretation of all we observe in differences in type in the course of syphilis, we shall have again to refer to in connection with the secondary symptoms. The virus of syphilis is received by persons having very different proclivities to disease. One may have a proclivity towards psoriasis, for instance; we do not know what that proclivity is, but we must take for granted that there is, in the disease which manifests itself in psoriasis, a constitutional predisposition. We know that that predisposition is more or less hereditary. So if I were to go through various affections of the skin I should have to make the same remark. I might make the general remark that patients differ very much

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in the liability for their tissues to inflame. The exanthemata, smallpox, and other diseases are very different in different individuals. We know that one individual may have hæmorrhagic variola, and another may have it severely confluent; others may have an exceedingly light visitation of the disease, while some may show two or three spots which it is very doubtful, if they are alone, whether, apart from the epidemic, they would have been considered as part of the disease. These differences in variola we must believe depend upon the idiosyncrasy of the patient, and not on the different virus. And so we apply our knowledge of the other exanthemata to syphilis, and we see that everything depends on the proclivities of the individual. In one person there may be a great tendency to ulceration and to inflammation in connection with the irritation of the specific virus of syphilis. In some cases the chancre may inflame, while in others the chancre will be quite quiet and as hard as gristle, not inflaming or ulcerating or suppurating in the least. There may be, of course —and about that we said enough last week,—a difficulty in that the virus of syphilis is often mixed with contagious pus elements and the parasitic elements in pus, which may be one reason why the chancre inflames in some cases and not in others. But I take it the general explanation is the proclivity of the individual to inflammation, and that goes through the whole of these secondary phenomena.

I will now just enumerate very briefly the chief of what we count as the secondary phenomena. We name it the exanthem stage, the outbreak stage, the stage of acute blood disease, when the virus has developed and is present in every particle of the blood and every portion of the body, and is liable to produce the disease here, there, and everywhere. Then we have the fever, which varies very much in different cases. In some severe cases the temperature may go up to 104°, and I have known a temperature of 105° on several successive evenings. Then there is the feeling of *malaise* and the general discomfort of being ill, which will vary with the tendency to fever. If there is much tendency to fever the patient will feel very ill. The whole febrile disturbance is due to the syphilitic virus. General *malaise* and fever, then, go together, and are generally proportionate. Then there may be bone-ache, what is called osteocopic pains. These pains may be merely an aching, or they may be attended by definite swelling. We do not call these swellings nodes in this secondary stage, although we might call them secondary nodes, and the others, which occur later, tertiary nodes. Sometimes there is a definite swelling on the scalp. These periosteal swellings in the secondary stage keep to the rule of all the rest of the secondary phenomena—that they are quite transitory and they never suppurate. They go away and leave no trace. Usually they last a very short time, and the occurrence of anything very obvious in the way of swellings under the periosteum in the secondary stage is very exceptional. Still, we see it every now and then. It is well to make it definite that there is in connection with these bone-aches of syphilis congestion and inflammation, which may go on to swelling, because that will give us some basis for explanation of the tertiary phenomena on the supposition that the tertiary phenomena are recrudescences of the disease in the site where it originally occurred. If these can occur in the secondary stage, we can understand how they can be exaggerated in the tertiary stage when we come to consider the true node.

Then we have the various eruptions which may occur. We use the term polymorphous in reference to syphilis, and I suspect we use it in different senses—some use it in one sense and some in another,—and that we are not always quite consistent with regard to what we mean by it. But I believe polymorphism, in the zoological sense, means the succession of different stages, not the occurrence of different forms of disease phenomena at the same stage. In syphilis, however, I believe that when we say an eruption is polymorphous as distinct from other eruptions, we do not so much mean that it is an erythema in one stage, a papular eruption in another, and squamous in another, and pustular or bullous in another; but that there may be at one and the same time, on the same patient's skin, various conditions of inflammations of the skin—that you may observe pustules, vesicles, and bullæ at the same time. At any rate, that is one sense in which we apply the term polymorphism to the syphilitic eruption, and it aids sometimes in the diagnosis of any individual case. We say if it is pustular in one case and papular in the other that it does not keep to any one type; therefore it is syphilitic, because it is polymorphous. The

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other use of the term would be in reference to the stages of the syphilitic eruption, which are very different at different periods. We insist over and over again that it is squamous at one time and erythematous at another, and so on. They are both true meanings with regard to the eruption of syphilis.

Syphilis imitates almost every eruption that you can imagine. I do not think you could mention any eruption on the human skin that has received a definite name, which may not have a close simulation amongst the phenomena of secondary syphilis or tertiary syphilis. We are accustomed to speak of special types of skin disease; the roseolous I need not say anything more about. That is the first eruption of syphilis, transitory, gone in a few weeks. Then there ensues a more persistent eruption, a papular one very often, and with that may occur pustules or papules or degeneration, and little sores may occur. And so you may have several types, either at the same time or in succession to each other.

I might mention syphilitic psoriasis as a type of syphilitic eruption. In this the papules are scaly, and there is no doubt that they are surmounted by silvery scales and desquamation; but the scales are not abundant, they are not heaped up, and they are not so silvery or so easily seen as in common psoriasis. It is a squamous eruption, but it does not come very close, so you are not very likely to make a mistake between syphilitic psoriasis and common psoriasis. But cases do occur in which it is exceedingly difficult to distinguish them. I saw not long ago a young lady who was covered with an eruption which I certainly took at first to be common psoriasis—white silvery scales heaped up in patches, which looked like ordinary psoriasis,—but in the end it turned out to be a case of the syphilitic form.

The difference between lichen and psoriasis is another matter. We have many lichenoid eruptions of syphilis. I mean any eruption involving chiefly hair-follicle, which is generally an induration around the hair-follicle, involving more or less all the structures which are related to it, such as sebaceous glands. The lichen papule is a little papule developed around the hair-follicle, and not confluent. Usually they are more or less scattered. The varieties of lichenous syphilis are very numerous, and the simulation to other forms of lichen may be very close.

There is in syphilis a very close simulation to variola itself, and I daresay some here have seen such cases. To those who have not seen such cases it may be astonishing to be told that the syphilitic exanthem is sometimes so like variola—hard, shotty papules in the first place—that it would deceive even the well-experienced dermatologist. Many syphilitic patients are every year sent into the smallpox hospitals as having variola, and are sometimes received there, though I do not say always with full acquiescence in the diagnosis; at any rate there has been sufficient doubt about it for the patient to be kept in until the matter is cleared up. I could not mention any fact more conclusive as an illustration of the fact that syphilitic eruptions may simulate all the eruptions which we know, than this close simulation to variola. I have been deceived by them, although I had written about the subject before, and therefore knew the fallacies connected with the matter. I was taken in by one patient who was carried out of a ship and so covered with eruption that I thought it was variola. But it proved to be syphilis, and it persisted until we cured it by antisyphilitic treatment. I have known several cases in which the patients have been sent into a smallpox hospital, and in which they have privately been isolated. One young gentleman was isolated in a garret and carbolic screens hung round the door, and he was kept there for a month. But the variola did not get better. I was asked to see him, and found it was a case of syphilis.

Then there is also a remarkable simulation to pemphigus, and in connection with this we have the best illustration of what I have asserted as the probable truth, that the modifications of eruption are due to the previously existing peculiarities in the patient's proclivities. I have suggested that we must suppose that some patients have a proclivity to psoriasis; and so we must suppose that there are amongst us patients who have a proclivity towards pemphigus. It is constitutional. I do not know why it is, because the patients appear to be in perfect health, and it is curable almost solely by giving arsenic and quinine in large doses. Arsenic is by far the best, and is almost a specific cure for ordinary pemphigus,—that is to say, non-specific pemphigus. Of this syphilitic form I have seen only two cases, but very well marked ones, in which the patients in the

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stage of secondary syphilis had an eruption of pemphigus. In each of these the patient was covered, and no one could diagnose between pemphigus and syphilitic pemphigus except by the history. The history was clear that the patient was in the secondary stage of syphilis. Now for the impression that it depended upon the peculiarities of the patient's constitution. Both these patients were very ill. Pemphigus involves great loss of health and strength; the patient is blistered all over his surface, and it reduces his strength very much. In each of these cases we feared the patient would die. Now mercury would not cure that; the eruption persisted in spite of it; we gave iodide of potassium, and the eruption came out more freely. The remedies which cured it were the combination of the specific for pemphigus and the specific for syphilis. We gave arsenic in full doses, and the pemphigus ceased; we gave mercury at the same time by pill, and got rid of the syphilitic element. In both cases ultimately, although they were long and trying cases, the patients recovered by the combined use of the two remedies I have mentioned. That illustrates that there probably are very rare cases in which we may suppose there is this peculiarity of the constitution which favours the development of pemphigus, and in which the manifestation of the syphilitic virus was modified, so that the eruption would not respond to the specifics for syphilis alone.

Then there are ulcerating eruptions, and the tendency to them. This tendency is very rarely shown in the very early stages of syphilitic eruptions, but it comes in a certain number of cases after a time; and if it is very pronounced, if all the places take the ulcerative form and form scabs, not only rupial scabs but extensive ulcerations, to this class of case the term syphilis maligna is given; and in these cases the health fails. In these cases of ulcerating syphilis, again, I invoke the supposition that it is the patient's constitution which makes the syphilis ulcerate, and nothing else.

With regard to malignant syphilis, I say, however much your patient is emaciated, however wretched and weak in health, however extensive may be the ulceration, still mercury is the thing; it will fatten the patient, and it will produce recovery even in the most apparently hopeless conditions.

I think I have mentioned to you some of the principal types which deviate from the rule in syphilis. I have said that in all these cases we are to suppose that the whole blood is infected, and in proof of that that in all these cases the eruption is quite symmetrical. Strip your patient, and you will find that it occurs on both limbs and on both sides of the trunk. When this was first stated, one of our leading authorities on syphilis expressed his incredulity as to its being specific. But one of the great improvements in the clinical examination of our patients of recent days has been that we strip our patients and have a look at the whole skin. Our forefathers used to be content to look at the arm, to let the patient lay bare a little bit of the eruption and to make a diagnosis from that; but now we follow the practice of making the patient strip. This gentleman I refer to, who was a leading authority on syphilis then, when I said that secondary syphilitic eruptions were symmetrical, expressed his doubt, because he had only been in the habit of looking at one part. But now we strip our patients by the dozen and examine them spot for spot; and we are quite sure of this, that during the secondary stage of syphilis the patient has the eruption all over him, and that the spots occur with very accurate bilateral symmetry.

The next question for our consideration is, how long is the duration of the secondary symptoms of the syphilitic eruption? Or perhaps I ought to hark back a little. I have gone into the details of the eruptions, but have said nothing about the mucous membranes. During the stage of the secondary eruption on the skin, the mucous membranes are all equally liable to suffer. There may be an eruption on the tongue, and the patient may have a syphilitic psoriasis on the tongue patch for patch the same on both sides.

There may be syphilitic ulcerations of the mouth, and you may have them, of course, very commonly on the tonsils, producing the well-known syphilitic sore throat. We used to be taught that the syphilitic sore throat consisted of kidney-shaped ulcers in the tonsils symmetrically placed. I believe John Hunter described those. I believe he did not accurately describe the indurated chancre, but he accurately described the condition of the tonsils. Very frequently in the secondary stage of syphilis the inflammation leaves the tonsils and extends on the sides of the palate—the soft palate,—and it is often characterised by the formation of white secre-

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tion—sometimes it is called snail-tracks—having a grey, glutinous appearance resembling the tracks made by a snail crawling over the surface. In many, however, you see a superficial ulceration extending widely about the throat, and characterised by grey edges, and not infrequently distinguished by a pedicle which you can pull off. There is some tendency towards the formation of a fine fibrous pedicle in syphilitic sore throat.

Then the eye may be affected. The iris may inflame, and nodules of lymph may occur, such as those which produce the papular eruption on the skin. And these nodules of lymph are generally coloured a red, rusty tint, the tint of the skin eruption in syphilis. I have not laid much stress on that, because everybody knows of it; it is the well-known coppery or raw ham colour, but it is not one which is invariably present. Sometimes these nodules are very conspicuous. Sometimes a large nodule will reach until it almost touches the posterior surface of the cornea. Iritis is a very rare event in syphilis, but it occurs every now and then. To suppose you can make any distinction between syphilis and other diseases by the eye phenomena would be nothing less than absurd, because the eye phenomena are so infrequent. I see a good deal of syphilis in private practice—I have not a hospital now,—but I have not seen a case of syphilitic iritis for years; and if you go to the hospitals you will find you will not see many. Sir William Laurence was, I believe, the first to observe inflammations of the iris in connection with syphilis, and it was he who first said there was such a thing as syphilitic iritis. He had the whole of St. Bartholomew's Hospital as his sphere of observation, and was also surgeon to the Moorfields Ophthalmic Hospital; and when he published his paper to prove that iritis did occur in connection with syphilis, he could only collect four cases, or about that number. That will give you an idea of its rarity. So you may see a great deal of syphilis and yet not see a case of iritis in connection with it. Its occurrence depends, I think, upon the peculiar proclivities of the individual, which may be partly a matter of race. I used to hold, and I hold still, that Scotchmen are much more liable to iritis than any other race I am acquainted with; the most severe cases of iritis I have seen have all been in Scotch people. Whilst the iris is affected you may also find an inflammation of the deeper parts of the eye; there may be present a pan-ophthalmitis, not suppurative, but an inflammation of the deeper structures, a pan-ophthalmitis in the sense of many of the eye structures being involved at the same time—retina, choroid, and iris. We owe to the memory of the late Dr. Jacobs this observation, that syphilitic iritis was not by any means exclusively an iritis; the iritis was the thing which could be most easily seen, but very often the patient's eye was damaged by inflammation of the deeper-seated structures. The eye affords us a remarkably good opportunity for observing the processes of disease in syphilis as well as other maladies. As regards syphilis, we may say that it affords us an opportunity of seeing that; and now that we have the ophthalmoscope we have no doubt that there is a syphilitic retinitis, during which the nerve-structures of the retina become hazy, slightly blood-stained, and pinkish. Also there may be inflammation of the choroid, and there may be an eruption on the choroid; a number of discrete little papules may form in the choroid, which can be very easily recognised by means of the ophthalmoscope, exactly similar to the condition on the patient's skin. So here we see, in places which are ordinarily beyond our view, that the syphilitic virus is exerting its influence, and probably it is doing so on all structures of the body; and what we see in the eye we may reasonably infer takes place in the membranes of the brain, and in the structure of the spinal cord and its membranes, as well as in other various parts of the body. With regard to the date, acute iritis is always a secondary symptom, and the same may be said of the retinitis and the choroiditis. We have other forms of choroiditis; there can be no doubt about that. Choroiditis disseminata, as its name implies, is a scattered choroiditis, just as you speak of dermatitis disseminata when the patient is spotted over. Choroiditis disseminata occurs in the secondary period. Serpiginous choroiditis does not occur as a disseminate condition; it is not largely scattered, but there are one or two large patches, and it creeps at its edges and spreads over large portions of the choroid.

Amongst other structures which we must mention as being very liable to be affected in this blood condition, when the whole blood is loaded with syphilitic poison, are the perivascular spaces,—that

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is to say, the delicate cellular tissue in the spaces around the blood-vessels, even around the most minute capillaries. We know all the capillaries are liable to be affected during the secondary stage of syphilis; sometimes more severely, sometimes less so. But there is risk that there may be an effusion into the perivascular spaces of any structure where such spaces exist; and the knowledge that all the arteries and other blood-vessels are liable to inflammatory effusion into the spaces around them is of extreme importance in interpreting the phenomena as regards the nervous system. Instead of saying we think the nervous elements are affected, or the nerve-cells, it very probably is an affection of the blood-vessels which supply them. There is no more important piece of modern pathology made out than that in the secondary stage of syphilis there is a liability to inflammation of the perivascular spaces, resulting in different things; sometimes in almost occlusion of a large patch of capillaries, sometimes pressure upon the trunk and an artery, and so on. Again, as regards the arteries, we recognise differences in various stages of syphilis. We merely assert that it does occur in the secondary stage. In the tertiary stage we may get large masses fused into the perivascular spaces. It is a general, wide-spread, slight effusion into the spaces surrounding the blood-vessels.

I have hinted that there is a tendency to spontaneous subsidence in secondary syphilis. Everyone will agree with that; no one sees a patient in whom the primary chancre persists indefinitely. Nowadays, of course, we have little opportunity of studying the natural history of syphilis uninfluenced by treatment with mercury. Still, the primary chancre has disappeared very quickly in very many cases, and where it lasted longest it did not last indefinitely. So we say of the secondary symptoms that they have all a tendency to spontaneous disappearance; they go away after a while, and completely. Exceptions occur in which they persist. I have said already that in our modern treatment of syphilis we attempt to anticipate the secondary stage. We give mercury as soon as we can be sure that the chancre is infective; we give mercury freely, and we hope never to see one of the secondary symptoms which have been described. The result is that in the great majority of cases, mercury having been begun early enough, no secondary symptoms are seen. But if syphilis has been allowed to develop uninterfered with by mercury, we shall still be able to witness the spontaneous disappearance or tendency to that. Of course since mercury had its repute there have been anti-mercurialists, those who thought it was better not to give mercury, but to rely upon the forces of Nature. I mention that fact now, not in any approval of it, but in proof of what I am saying, that if you do leave them alone they do get well. How long would it last? My impression is that a majority of patients who have secondary syphilis, if they had no treatment, would be rid of the symptoms within six months. Another considerable proportion of those who were not so fortunate would be free of them in the course of a year; and a great many, even in modern practice, get well spontaneously. I often see patients with tertiary symptoms long periods after their symptoms, when one finds they have not taken anything, and they scarcely had any secondary symptoms which were noticeable. But, however severe they are, I believe in the whole of the cases there would be a complete disappearance of all the symptoms of the disease. I have alluded to symmetry, and I have said enough as to its meaning. If a disease is non-symmetrical you may assume that the blood can be diseased still, but certainly the blood is not the sole agent in its production. Another general fact with regard to secondary eruptions I will appeal to, and that is that they never creep at their edges—they never spread. Syphilitic rupia, as I have said, leaves the round shilling scar; it does not leave a great irregular scar; it does not spread in crescents or like a horseshoe, as in tertiary syphilis; it is not infective at its edge. I take it that the explanation of that is that all the tissues have got the poison alike, so that there is no tendency for this particular local inflammation to spread into the tissues with which it is in continuity, because these tissues are already diseased; they have got the poison. In the later stage, in the tertiary affections, the poison will have died out of the blood and the tissues, and is no longer active. So that secondary syphilides disappear spontaneously, and they are at no period serpiginous; they do not creep at the edges; you will find but very little evidence of creeping.

As to when the secondary stage ends, we have to discuss that in social life in several ways. We

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say that in the whole of the secondary stage there is risk of contagion. If the patient who was in this stage were vaccinated and lymph were taken, that lymph would run the risk of conveying syphilis. The secretion from any sore he might have would run the risk of conveying syphilis. Probably, as the case goes on, the further the distance from the period of the secondary phenomena the less active is the poison, and the less abundant are the elements of the syphilitic poison in the blood. But the risk is still there. And so far as the question of marriage is concerned, we say that the risk of contagion lasts two years. In saying that we wish to be on the safe side, and we are probably giving a period which is much in excess of the real duration of the poison in the great majority of cases; but there is no doubt whatever that in some cases the patient is capable of producing contagion as long as that. Some say he is liable to transmit it at a much longer period than that. There is a popular notion that for six or eight years a patient may affect his wife. My syphilitic experience is against that. I will not be too dogmatic, but I allow my patients to marry if two full years have passed from the beginning of the syphilis. Of course there must be an absence of symptoms for a year, but I have allowed hundreds of people to marry under such conditions, and I have never yet had cause to regret it; I have never seen a syphilitic infant born in a marriage in which I had permitted that marriage. I have been told of such things, but in all the cases in which I have had an opportunity of examination I have come to the conclusion that there was no such thing, and that it was not a syphilitic child. The patient goes away full of anxiety; he goes and tells his doctor that he has had syphilis. The doctor looks at the baby, and so there will be a false diagnosis. I have always asked to see the child if I have been told this, and I have never yet had a conclusive instance of a syphilitic child being produced in a marriage which I have permitted after two years have elapsed. I have known contagion occur in cases where patients have married without my permission just within the two years—a few months within,—but I still keep to my rule, and I have not had any reason to regret it.

The duration and the contagion stage also comes into prominence in reference to legislation for the so-called suppression of syphilis,—that is, compulsorily secluding prostitutes. There is not any sort of use in doing it, for the period of contagion lasts so long. If you took a prostitute into hospital to be cured, it would be necessary to keep her under observation and secluded from the risk of contagion for two years. That strikes at the root of all legislation on the subject. You cannot do that. But in saying that it strikes at the root of legislation I do not think it strikes at the root of certain other measures which might do a great deal for the diminution of the prevalence of syphilis. I am no advocate of them; I do not think they would attain their end, and I think they would be injurious to the morality of the community. But if opportunities could be very much increased to favour women affected with syphilis coming early under treatment, and remaining under it for a considerable time (although I admit it needs two years to be perfectly safe), I believe they would be secure under good mercurial treatment at six months; and it would be a great safeguard if they could be kept under treatment so long, for in the great majority of cases the cure would be practically complete by that time. Our forefathers used to allow their patients to marry as soon as ever their secondary symptoms had disappeared. I have seen a certificate, signed by the then president of the College of Surgeons, given to the patient three or four months after his syphilis, certifying that all symptoms had disappeared and that he was justified in marrying. Nowadays we should not think of such a thing as that. Still, I think that probably our forefathers were very nearly safe, though they were not quite. We want to be quite safe. Taking the disease known as yaws in the West Indies to be what I think most of us believe is nothing but syphilis in negroes, they supply us with very interesting data with regard to the disappearance of the secondary stage. In the sugar plantations in the West Indies they took the negro affected with yaws into a hospital which was established on the estate, and into which all the yaws patients were admitted, and they were kept in the hospital for four or six months until the symptoms disappeared. The patients were not usually treated with mercury; they were simply kept there until they got well. And the negro who had had yaws and gone through with it was thought to be worth more than two or three others, because he was not then liable to a secondary attack.

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We next come to the question of second infections, and we come to ask what second affections prove. I have said the negro increased in value after he had gone through his syphilis. We know in England that a patient with syphilis is liable to second attacks. In England it was thought people were not liable to secondary attacks of syphilis, but now we see such cases, and I take it that the occurrence of second attacks may be taken as proof that the virus has died out of the system; it proves that the patient has practically got well, got rid of the risk of tertiary symptoms, that he is rid of it as an infective disease.

Next I have to speak a few words as to cases which persist. Fortunately they are very few, but there are a few in which the syphilitic eruption resists treatment. I have mentioned almost the only one that I have encountered, but there are others in which more or less the eruption resists treatment; in which it may all but disappear, leaving very slight traces, but still you cannot quite get rid of it. There are cases in which the mouth and tongue will continue to be sore, and in which there will continue to be eruptions on the palate. These very often are referable to the habits of the individual. Smoking, for instance, constantly keeps up syphilitic symptoms. Some of them depend upon a peculiar liability to what is known as herpes, which appears to be induced by syphilis. Herpes will appear over and over again in the mouth or on the face, and on any part of the mucous membranes where herpes is common, after an attack of syphilis, and is not amenable to mercury. It is then a nervous symptom, and may often lead you to think that syphilis has persisted in the patient when it has not. It is not then a blood disease any longer; it is an herpetic manifestation, and is connected with the nervous system.

MAHU has obtained distinct results in the treatment of ulcerating epitheliomas by simple application of bandages saturated with adrenalin. It lessened the hæmorrhage, relieved pain, and there followed a rapid improvement in the general health. He is not prepared to state that this substance will cure cancer, but it has appeared to arrest the progress of certain growths for a considerable time.—*Medicine*, July, 1903.

SUCCESSFUL TREATMENT OF A CASE OF SPASMODIC ASTHMA.

Under the care of E. C. GARMAN, L.R.C.P.Lond., and A. H. JACOB, L.R.C.S.I.

THE treatment of asthma by drugs is so often merely palliative that the following case seems worthy to be placed on record as illustrating an alternative method which has, in this instance, led to a gratifying result.

R. C. G—, æt. 39 years, married, a stockman and farm labourer, first came under observation in 1894, and each succeeding winter found him the subject of winter cough and attacks of asthma of more or less severity. He said that he had been treated for asthma each winter since 1891. He stated that in the autumn of 1890 he was working one day against time to get some waggon-loads of corn cleared from a barn before nightfall. He felt that he was overtaxing his strength, and was so exhausted that he walked home with difficulty. Up to this time he had enjoyed perfect health, but for some months afterwards he did not feel up to his work, and complained of a constant dragging pain referred to the xiphisternum. The attacks above mentioned were treated by the ordinary methods, viz. belladonna, Pot. Iod., Æther. Sulph., burning nitre-paper, Himrod's asthma cure, etc. These gave temporary relief, but in the main the condition was unaltered.

When we saw the patient in January, 1901, he had been confined to his bedroom with a severe attack of asthma (since the previous Boxing Day, *or rather more than a week*). This he attributed to alcoholic indiscretion on Christmas Day. He was propped up in bed, and all his attention was occupied in struggling for breath. He had only had fitful snatches of sleep for some days, and was quite exhausted. His face was of a dusky hue and his conjunctivæ were suffused. On examination his respiration was found to be abdominal, and his chest was very emphysematous. At the level of the nipple the chest *excursion* was *nil*. Expiration was prolonged, and there was a great quantity of frothy expectoration. The heart-sounds could hardly be heard on account of the sonorous moist râles all over the chest. Percussion gave no definite results on account of the emphysema, and the apex-beat was very diffuse. There were no

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audible murmurs. The radial pulse was 124, soft and irregular, but the artery wall was normal. The patient's extremities were cold and clammy, and his tongue thickly furred. He complained of flatulent dyspepsia and nausea; his bowels had not acted for two days, and the urine was scanty and loaded with urates, but contained no albumen or sugar. The temperature in the axilla was 99·2° F.

Treatment.—A sedative expectorate mixture, with ethereal tincture of lobelia and a calomel purge, gave the patient some relief, and the severity of the symptoms had somewhat abated on our second visit. Belladonna, stramonium cigarettes, nitro-glycerine, morphine hydrochlorate, antipyrin, and "green mountain cure" were tried in succession to relieve the bronchial spasm. Of these, belladonna and morphia were badly borne, and nitro-glycerine was useless; "green mountain cure" was the most successful, and the patient had frequent recourse to inhalation when the spasm got worse. At the end of six weeks' treatment the patient was better in general health and was getting more sleep. He had not, however, been able to resume the recumbent posture. The dyspepsia required very careful dieting, and he occasionally vomited his food if given in any quantity. His bowels never acted without a purge, which usually gave temporary relief to his chest symptoms.

We now decided to give the Kingscote treatment (*vide* 'Asthma,' by Ernest Kingscote, M.B., published by Henry G. Glaisher, Wigmore Street, London, W.) a trial. These methods are devised on the assumption that "the cause or causes of asthma must be sought for in a lesion or lesions of an organ or organs supplied by the vagus, or sympathetic nervous systems. Treatment, remove the cause or causes." Kingscote states that a dilated heart which may press on the vagi in the thorax is an almost constant accompaniment of most cases of asthma, and is veiled by emphysema, and that both of these conditions are produced by asthma. We therefore examined the heart with a Kingscote pleximeter, which brings out accurately the deep cardiac percussion-note through the layer of emphysematous lung, and found that the left heart extended nearly to the mid-axillary line and the right to about a finger's breadth beyond the right border of the sternum. No murmurs could be heard, but the ventricles were asynchronous. The pulse at the wrist counted 120, was of low tension, and irregular. Respiration was laboured, but the expectoration much less profuse. The liver was large, and tender on percussion. Four gentle Schott's resisted exercises were then administered alternately with each arm, and an interval of two minutes allowed between each. The patient found these very hard work, but after their termination the pulse was only 116 and of fuller tension. These exercises were continued each morning for a month, gradually increasing in number and force until the patient could bear most of those recommended by Professor Schott, of Nauheim—namely, 16.

With one or two exceptions, when there was dyspeptic trouble, the pulse showed improvement in quality and diminution in rate after each series of exercises until the mean pulse-rate was 92. On the expiration of the month the general improvement was remarkable. The patient had lost the anxious, livid expression, and was able to walk about slowly on the flat, but found it very trying to get upstairs. His heart had receded to a point just outside the nipple line, while the area of liver dulness was much less and all tenderness had disappeared, but the emphysema was undiminished. The tongue remained much coated, and the food was returned from the stomach on the slightest indiscretion of diet. The patient was now instructed in the special breathing exercises recommended by Kingscote for the reduction of emphysema, and at the same time his wife was taught how to administer Schott's resisted exercises, and was told to discontinue them on the slightest sign of embarrassment on the part of the patient. This practice is not, of course, to be recommended, but seemed to be the best alternative as we were unable to continue the exercises ourselves. During the next month the patient had two bad attacks of spasmodic asthma, but resumed his improved condition after the lapse of a few days. As the state of the heart did not seem to be the only cause of these attacks, we made an examination of the stomach, which was found to be in a state of myasthenia and greatly dilated. This, no doubt, accounted for the chronic dyspepsia, and had been overlooked by us on the supposition that the congested liver was sufficient cause for the trouble. This condition might well be the cause of the two asthmatic spasms when one remembers the distribution of

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the vagus of the stomach. The patient was therefore put on a diet of peptonised milk, plasmon, and somatose, and his stomach was washed out with warm water every second morning. This procedure gave speedy relief to the dyspepsia, and the tongue cleaned rapidly. After a little time the patient acquired the habit of passing the stomach-tube himself whenever he felt any flatulence or pressure in the epigastrium. The diet was gradually altered until he could with impunity take fish, short-fibred meat, toast, grated cheese; and in a few weeks he was allowed an easily digested full diet. The patient's improvement was now steady; he was slowly approximating to his normal life, could walk upstairs with freedom, and for two miles or more on the flat. In fact he might have been pronounced convalescent were it not that his rest was disturbed every night by a short attack of asthma, which only yielded to inhalation of "green mountain cure" after about half an hour. We assumed this to be a pure habit-spasm (*vide* "Causes of Delay in the Cure of Asthma" in Kingscote's book) due to hyperæsthesia of the vagi and habit-spasm of the bronchial muscles, so the patient was sent for a fortnight to the seaside in order to try and break the habit, with complete success. Shortly after his return we examined him finally, preparatory to his resuming work. The heart was normal in size, and the apex-beat could be felt within the nipple line. The emphysema was manifestly less, and the chest excursion was 1½ inches, whereas before the exhibition of the breathing exercises it was *nil*, and the breathing entirely abdominal. The dilatation of the stomach had disappeared, and the bowels, so constipated before, now worked with regularity. The pulse was firm and regular, and counted 84. A Largiarder exerciser was purchased for him, and he was instructed in its use and told to continue for at least six months, in addition to the breathing exercises. The patient was next seen on March 13th, 1903, when the emphysema had entirely disappeared, there being no hyper-resonance, the chest excursion having increased to 3½ inches, and the breathing being thoracic. The heart margin could now be percussed out with the finger, and there had been no return of asthma. The patient had resumed full work as a corn-carrier and labourer, etc.; in fact, on March 8th he had lifted a sack of corn weighing two hundred-weight into a waggon without untoward result. That a similar result may not be attained in every case of asthma can only be proved by those who have more experience with the methods than we; but no treatment which offers a fair chance of relief from this terrible scourge without danger, if properly carried out, is unworthy of a trial. The interesting features in this case seem to be that the sustained causes of vagal irritation were lesions of the heart, the lungs, the stomach, and the liver, and it was not until all these lesions had been severally treated and cured that the bronchial spasm disappeared. The initial cause in this case seems to have been a dilatation of the heart through the strain followed by cardiac dyspnœa, emphysema, myasthenia and dilatation of the stomach, and congestion and enlargement of the liver. The asthma may have been produced by any or all of these lesions. Another interesting point is the habit-spasm of the bronchial muscles akin to trade-spasm, which was relieved by reassuring the patient of its temporary character, and by complete change of air.

We have now watched him for the period of twelve months; he is able to resume the full work of a farm labourer without inconvenience or discomfort.

Phototherapy in Gynæcology.—Makaveyeff has invented an appliance which is a combination of a vaginal tubular speculum with an electric incandescent lamp that can be used for transmitting the effect of light upon the internal genitals in women. Phototherapy, according to the author, has given very good results, especially in the chronic diseases of the uterus and appendages accompanied by neuralgic pains. Light may be applied to the internal female genitals through the abdomen by means of the ordinary incandescent lamp with a suitable reflector, as electric light does not affect the surface of the body alone, but penetrates into the deeper tissues. The author has devised two types of apparatus in which small lamps can be introduced directly into the vagina. They are of such size that they do not give off too much heat.—*New York Medical Journal* and *Philadelphia Medical Journal*, July 4th, 1903.

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A CLINICAL LECTURE ON MALIGNANT DISEASE OF THE LARYNX.

Delivered at Westminster Hospital

By P. R. W. de SANTI, F.R.C.S.,

Aural Surgeon and Surgeon for Diseases of the Throat to the Hospital.

I WANT first of all to show you two throat cases (a third one was to have been here but has not come), and then to say a few words about malignant disease of the larynx. The first case is a man aged fifty, and he came to me about a couple of months ago with a history of pain in his tonsils, the pain shooting up to the ear; it was a radiating pain. He also had a marked swelling on the left side of the neck. He did not give a history of any trouble in the shape of swallowing, and he was not hoarse, but he had a certain amount of cough. The chief thing he complained of was pain, and he referred that to the ears and the region of the tonsils. There was no history or any evidence of syphilitic trouble. He had had no hæmorrhage from the larynx. He had recently been losing weight. His work is that of an outside porter; he uses his voice a good deal, and lives in a dusty and smoky atmosphere. The first thing to do is to look at his pharynx. We found that there was nothing to be seen in that region at all; his tonsils were perfectly clear, and there was nothing to account for the symptoms he felt in that region. On looking at the larynx one found that the whole of the epiglottis was greatly enlarged, nodulated, fixed, and in a condition of ulceration. After cocainising him and getting the pharynx fairly insensitive and asking him to take deep breaths, one was able to get a view past the epiglottis of the larynx proper, and it could be seen that the ulceration had extended from the epiglottis to the left aryepiglottic fold, and that

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the left side of the larynx was involved in the ulceration. The nodular condition of the parts, the age of the patient, the ulceration, the fœtor, the immobility of the parts, and the condition of the glands in the neck, which were hard, fixed, and deep-seated under the sterno-mastoid, made it practically certain that the man was sufferfng from malignant disease, the most common form of malignant disease at this age and in this situation being epithelioma. You will not be able to see very much more than the epiglottis, because the epiglottis is so much enlarged and ulcerated that it hides the larynx proper, even though you get him to phonate. The swelling extends over to the left of the larynx, and involves the left part of the interior of his throat. I invite you to feel his mass of enlarged glands in the neck.

There were two other cases I wanted particularly to show you. One of them, who I am sorry to say has not come, was a younger man with a growth on his vocal cord which simulated malignant disease. But here is another man, aged sixty, who came up complaining of shortness of breath, a good deal of cough, difficulty in breathing, some nasal stenosis, and loss of voice and hoarseness. He complains also of pain in and around the throat. From the history of the case and the age of the patient one thought the man might be suffering from malignant disease. But it is not so. It is a very excellent example of fixity of one cord, the right one. On inspection you will see that that cord does not move at all. Of course the history is one which might make one suspicious of malignant disease, especially as the right cord was found to be immobile, an early symptom in malignant disease. But in this man it is easy to satisfy ourselves that there is no malignant disease. It is true he has a cord which is fixed, but that fixity is not due to the presence of any growth. The cord is normal in appearance; there is no tumefaction or redness in connection with the cord. He has had gout and rheumatism very badly, and the diagnosis which has been made is that he has got ankylosis of his crico-arytænoid articulation on the right side, and it is that which is producing the fixity of one cord.

I am sorry I have not one or two more cases to show you by way of contrast, cases of innocent growths of the larynx. However, you have seen one case which is malignant, and another which simulated malignant disease. The most important thing to recognise in regard to malignant disease of the larynx is that it is not anything like as uncommon as many people think. A good many of these cases are seen now, and this is due to their being better recognised and more readily diagnosed. In olden days they were seldom diagnosed, and therefore naturally thought to be very rare. A great deal of work on the subject has been done in England, particularly by Semon and Butlin; and owing to their researches we are able to give a fair account of these cases, as to their symptoms, their diagnosis, and their treatment and prognosis.

The first thing that should be remembered in cases of malignant disease of the larynx is the classification of these growths into intrinsic and extrinsic varieties; this is very important both as to prognosis and treatment. Krishaber divided them into two classes. The intrinsic variety includes those which grow from the true or false vocal cords, the ventricles of Morgagni, or from below the vocal cords, in the infra-glottic region. Sarcoma or carcinoma growing from those parts is styled intrinsic malignant disease. The other variety, called extrinsic, you have seen an illustration of to-day. In this kind the growth starts from the epiglottis (principally), from the ary-epiglottic folds, the hyoid fossa, and the inter-arytænoid space. You will ask what is the use of this division, seeing that the disease in itself is the same? The great importance of it is that in the extrinsic variety you get very early glandular infiltration. In the case of the man you have seen this afternoon you will have noticed that the history of his trouble was only a short one, yet there was a very large mass of glands in the neck on the left side. He has now got a similar trouble commencing on the right side, and it has been pointed out by Krishaber, and has been practically supported by other observers since, that in the extrinsic varieties glands in the neighbourhood early become implicated; and therefore the prognosis is very grave as regards removal of the disease. In the intrinsic varieties, which grow from the true or false vocal cords, etc., the glands for some reason do not tend to become enlarged for some time. I have seen a good number of these cases, and as a matter of fact I am at present working at the subject.

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One of the great difficulties is to make out why there should be this difference in the behaviour of the glands in a disease which is exactly similar in character, and which only differs in the fact that it attacks one part in one case and another part in another case. One has examined these cases from a pathological point of view, and it comes to this, that the lymphatics themselves are distributed very freely all over the larynx, both the interior and the exterior, and if one makes an injection with mercury into the lymphatic vessels one can often find, if it is done with care, that all the small radicles get filled with the mercury, and that there is an extraordinary meshwork of lymphatics all over the interior and exterior of the larynx. But there is this to be said: your injection is more difficult to make the nearer you get to the true or the false vocal cords; that is to say, the lymphatic channels get smaller and smaller as one gets towards the true and false vocal cords. On the epiglottis and the aryepiglottic folds the lymphatic channels fill very easily. The reason is that they are larger than the small radicles which are found about the true and false vocal cords. I think a reason why the glands are not enlarged in intrinsic carcinoma is because there is a mechanical difficulty to the entry of either the cancer cell or juice which causes glandular infiltration. I believe it is the cell itself, and that these cells cannot get into the smaller radicles, and that therefore the glands do not become infected in the early stages of intrinsic carcinoma. In the extrinsic variety the vessels are much larger, especially on the epiglottis, and therefore the cells can get into them, and so there is an early implication of the glands. This is a most important point, because it deals with the prognosis and treatment of the cases. If you have a case like this man, who came to me two months ago with this mass of glands in his neck due to an extensive extrinsic carcinoma of the epiglottis, you may know well that no operation could possibly be of any benefit to him. I told him so, and he has not been subjected to any operation. No operation, extensive enough to get rid of the whole disease, could possibly be done in this case; the disease is beyond safe or efficient operative measures. The deep-seated glands are enlarged on both sides of the neck; he has got implication of the glottis and of the larynx proper, and I believe the disease is beginning to involve the base of the tongue. To attempt to remove all those parts would be unjustifiable. Though it is only two months since he began to complain of trouble he is already too far gone for operation to be of any use. Thus you will see the prognosis in extrinsic carcinoma of the larynx is very grave indeed.

As a contrast to this case I may tell you that an oldish man came to me some time ago, in rather feeble health, with a history of hoarseness, and considerable difficulty in breathing, and pain radiating to the ear. On looking at his larynx I found he had a very typical epithelioma of one vocal cord. The cord presented an appearance which is very characteristic; it was swollen and infiltrated, with a distinct neoplasm growing from its posterior third. The cord itself was fixed by infiltration. The man had no enlarged glands at all, and one was able to quite easily diagnose an intrinsic epithelioma of the larynx. This was a man who had not had the disease very long. He was between sixty and seventy years of age, and only one half of the larynx was affected; it was limited to the interior. There was no glandular infiltration. His was an intrinsic carcinoma which, if subjected to operation, should have given good results. I operated upon him, intending to do the comparatively small operation of thyrotomy, but it turned out to be necessary to do much more, namely, removal of one half of the larynx. He should have made a good recovery, but in a few days' time he could not swallow easily. He was very intolerant of nutrient enemata, and he died a week afterwards of syncope. In his case a post-mortem was made, and I had a very careful examination made of the glands in the neighbourhood, which were not felt to be enlarged during life. Post mortem these glands were removed and examined microscopically, and were found to be quite free from secondary growths. That bears out what I have said, that in intrinsic malignant disease the glands are not enlarged until late, whereas in the extrinsic they are enlarged very early, and the prognosis is so much the worse.

We next come to the symptoms and diagnosis of malignant disease of the larynx. I am only talking to-day about carcinoma; sarcoma has a few characteristics, which I shall not have time to go into. Carcinoma of the larynx is a condition of affairs which must, if you are going to have a good result, be diagnosed very early. We will

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first of all take the intrinsic variety. In these cases the patient generally comes to you with a history of hoarseness. That is the first and usually the most important symptom. Generally the patient is over sixty years of age; as a rule the disease does not occur in young people. There is often pain, which radiates to one or other ear, or to both ears. This is another very characteristic symptom. Later on, as the disease spreads, you are apt to find a certain amount of cough, a certain amount of secretion, and in the later stages a great deal of secretion, and also a certain amount of blood-stained sputum. In later cases, again, when the disease has spread there may be almost fatal hæmorrhage from the bleeding of neighbouring vessels. If you examine the patient with the laryngoscope for this condition, you will find in the intrinsic variety that the disease usually presents itself in one of two ways. First of all, it is most common to find in the intrinsic variety the true vocal cords affected. Some authorities contradict this, and state that the false vocal cords are more often affected than the true. But as far as statistics are concerned—and they are extensive now—undoubtedly the true vocal cords are more commonly affected than the false, and the disease may present itself either as a general swelling and redness of one cord with tumefaction and infiltration, or else it may present itself, and very often does, as a deep infiltrating growth situated on the posterior part of the true vocal cord, the growth being characterised by a somewhat cauliflower-like appearance, very much like a wart, but broader at the base, with the papillæ nothing like so long as in papilloma of the larynx; by its solitary character, and—this is the most important point of all from a diagnostic point of view—the limitation of movement of the part. These are the symptoms seen in early stages—either tumefaction generally of the vocal cord, or else a growth which is more or less like a papilloma. In both cases the great characteristic symptom is the immobility of the cord or limitation of movement of it. One may go so far as to say that if you get a patient presenting himself to you complaining of hoarseness and nothing else except a little pain, who is over sixty, and in whom when you examine the larynx you find one cord distinctly fixed on attempts at phonation, you have to be very suspicious of its being malignant disease. You must exclude other causes of hoarseness; but, at the same time, when you get chronic laryngitis which causes hoarseness, the result is for both vocal cords to be affected. If we take extrinsic carcinoma you will find that the epiglottis is the part most frequently attacked, as in the man you have seen, and the epiglottis will appear large and swollen and fixed, is infiltrated, and may present one or more nodules on its surface. On examination with the laryngoscope you find ulceration present in most cases. Ulcers—and in the later stages ulceration is exceedingly common in connection with malignant disease, whether on the inside of the larynx or the outside—are characterised by being of the usual malignant type—ragged, somewhat depressed ulcers, with heaped-up edges, crateriform, as it is called, the edges looking everted. And if by any chance you can get the finger back and feel the part, the diseased structure feels like epithelioma anywhere else; it is broken down, the fœtor is considerable, and the discharge sanious. In extrinsic carcinoma you will have, as corroborative evidence, the condition of the glands. Supposing you have a case presenting these symptoms, I do not think there should be much difficulty in the diagnosis. It is in the early cases that the diagnosis of malignant disease is so difficult, and in which it is so important to make it, because if you are going to do anything operative the earlier you carry it out the better for the patient. If you get a case with limitation of movement of the cords and hoarseness, with a distinct swelling of the whole cord, or part of it, I should advise a thorough cocainisation of the part and removal of a portion of the infiltrated part, with the view of its being subjected to examination by a skilled pathologist. That is the proper treatment in cases of doubt. If a piece is carefully snipped off by an experienced man and it is carefully examined, you will probably be able to get conclusive evidence one way or the other. If you find there is no evidence of malignant disease, then you may watch the case for some time, to see how it goes on. If there is any evidence of its being epithelioma the sooner you operate the better.

The diagnosis of malignant disease is not always easy. One has to differentiate between malignant disease and chronic laryngitis, and between malignant disease and pachydermia laryngis, and between that and a benign tumour of the larynx.

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Some have confounded malignant disease with lupus of the pharynx; and very often there is a great deal of difficulty in diagnosing between cases of malignant disease and syphilitic affections of the larynx. Again, you sometimes find, as in this man, some form of paralysis of the larynx which at first simulates malignant disease in the early stage. If you take the diagnosis between malignant disease and chronic laryngitis you will find generally that there has been some distinct cause for the chronic laryngitis; the patient may have used his voice a great deal in shouting, or he may have been subjected to a great deal of general irritation in the shape of dust. You will find there is a long history of hoarseness, and that it has existed for a longer time than malignant disease could have done without showing more signs; moreover in this disease there are times when the patient feels better, and others when he feels worse. Pain radiating to the ear is usually absent in cases of chronic laryngitis, and there is no enlargement of the glands. On looking at the larynx with the laryngoscope, as I said before, in chronic laryngitis there is a general redness, as a rule, of both vocal cords; the cords may be swollen, and they are uniformly red, and there are also present other symptoms of chronic irritation of the larynx. There is no tumour, or fixity, or impairment of mobility, but you have perfectly freely moving cords, uniformly red, and the absence of any actual tumour formation about the larynx. These evidences make it certain that you have to deal with an ordinary case of chronic laryngitis, and not with a case of malignant disease.

Then we come to tubercle. There, again, as a rule, it is quite easy to make your diagnosis. In tubercular laryngitis you get a great deal of tumour formation. Tubercular nodules may and do form in tubercular laryngitis, but these nodules are generally multiple, and both cords are generally affected, the interarytænoid space is nearly always swollen, and the pain is greater than it is in malignant disease, and does not shoot up to the ear as it does in the latter. The tubercular nodules are very liable to break down and form ulcers, and these ulcers, when they are formed, are always multiple, as against the one single ulcer of malignant disease. These tubercular ulcers are scattered on both cords and in the interarytænoid space, and they are shallow, and there is nothing like the raised everted edges which are seen in malignant disease. Another thing which will strike you in tubercular cases is the pallor of the pharynx and larynx, except in the parts affected by the tubercular infiltration. Again, the parts invaded by tubercle are often freely movable, whereas in malignant disease there is limitation of movement. Moreover tubercular disease occurs in much younger people than does malignant growth, and one can generally detect symptoms of tubercular mischief in other parts of the body, such as the lungs, and examination of the expectoration will generally reveal the presence of tubercle bacilli. Sometimes the cases are complicated, and there are on record cases of mixed tubercular and malignant infection, but these are very rare.

To diagnose malignant disease from syphilis is sometimes quite easy, while at other times it is extremely difficult. I had a case the other day which I believed to be malignant disease, and which I had into the hospital with the view of doing some operation. He was seen by two or three of my colleagues who had done throat work, and whose opinion was therefore of value, and they all differed in their opinion; some thought it was malignant and others regarded it as syphilitic. Though the man was put upon iodide of potassium he did not improve, and I fancy that the case was malignant; however, I have lost sight of him now. But the disease was extensive, and it would not have been suitable for operation. In the usual way you can tell whether a case is one of syphilitic disease or of malignant disease; both may occur at the same time in life, though it is usual for syphilis to be seen rather earlier, namely, at between forty and fifty years of age, whereas malignant disease is more common between sixty and seventy. Of course, the syphilitic manifestation is a tertiary one, and the disease is characterised by the amount of ulceration which is present from the first, by the extent to which, and the rapidity with which it spreads. If there is a gumma forming in the larynx it is characterised by the usual appearance of a gumma, a punched-out, somewhat round or oval ulcer, with a wet chamois leather base to it, no heaping-up of the edges, but a deep depressed ulcer. You will generally find there is more than one ulcer, and that the ulcers are apt to unite and form one large mass, which causes considerable destruction of the larynx, and

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eventually may lead to necrosis of the cartilages and to bad stenosis. The parts affected are generally the same as in malignant disease, and when there is infiltration of the parts the extent over which there is infiltration and the absence of tumour formation are points helping the question of diagnosis. The glands in the neck do not become involved. The result of treatment is quite easily seen by giving large doses of iodide of potassium—twenty-five to thirty grains three times a day will soon cause an improvement in the condition of a very bad case of tertiary syphilitic disease of the larynx, whereas in malignant disease it generally has but little if any effect. Difficulty in differentiating it from lupus should never arise. Lupus occurs invariably in young people from sixteen or seventeen to thirty years of age, and it is extremely slow in its course. There is a regular nodular appearance, which affects the epiglottis and other parts, and very often there is a similar condition in the face. There should be no difficulty in diagnosis.

There is another disease that I have mentioned which sometimes causes difficulty in diagnosis, and that is pachydermia laryngis. In a man who should have come here to-day there was a question whether he had pachydermia laryngis or malignant disease. His age was forty-five, which is early for malignant disease. I took off a piece of the growth with endolaryngeal forceps. It was not malignant; it was called a papillary growth. It turned out to be a case of pachydermia laryngis. In this man's case there was a growth on the left vocal cord, which was thickened at the base, nodular, and somewhat like a papilloma; and on the opposite side, corresponding to the nodular growth on the cord, was seen at a later period a depression. When the man phonated the two cords came well together, and the enlargement of the left vocal cord fitted into the depression on the right one. Pachydermia laryngis means chronic laryngitis which has gone on to the formation of a distinct papillary growth. The cord was congested. The depression on one cord was not visible when he first came, and as there was simply the growth on the other cord it was thought likely to be a case of malignant disease. The appearance of the growth was slightly nodular, with apparently no infiltration at its base and no tendency to break down into an ulcer. It looked like a mass of layers of epithelium heaped up. When this cup-like depression formed on the other side one found it was a case of pachydermia laryngis, and he is now being treated for that. The general appearance of the growth was suggestive of malignant disease, but there was free movement of the cord. There was also a history of alcohol, which, with the other facts, made it certain the disease was not malignant. It is important to remember in this connection that if the patient does go on smoking and drinking, the condition may eventually lead to the formation of carcinoma. Just as in old days carcinoma of the scrotum was traceable to the constant irritation of the soot at the part, so the chronic irritation produced by alcohol on the throat may lead to malignant disease there.

Now we come to the prognosis. There is very little to say about the prognosis of malignant disease of the larynx. The prognosis of malignant disease anywhere in the body is about as bad as it can be. There is much talk about operations for malignant disease now being successfully done. For many years I had to perform operations in general as well as in special throat surgery, and as far as one knows recurrence is the rule in almost every variety of malignant disease. People say they have cured a patient of malignant disease if he is kept free from recurrence for three years. If you remove a cancer of the breast, and the patient is free from any recurrence in the glands or elsewhere three years afterwards, it is the usual thing to say that that patient is cured of the disease. However, if you trace these cases later you will find that the rule is recurrence. So the prognosis in malignant disease is bad anywhere, and we must compare the results in the larynx with those elsewhere. Up to within the last few years malignant disease of the larynx was a condition which almost invariably ended fatally; very little could be done for it. In former days the favourite thing was endolaryngeal operation. The patient was cocainised, and a pair of cutting forceps was put in with the aid of the laryngoscope and the growth seized and removed. That is absolutely condemned now, for such a procedure results in a more rapid growth afterwards. Nowadays a piece is taken out for microscopical purposes so as to clinch the diagnosis.

What are the measures which are open to us in

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any given case of malignant disease? The operations which are practised nowadays for malignant disease are, first of all, thyrotomy or laryngo-fissure; secondly, removal of one half of the larynx itself; and thirdly, removal of the whole of the larynx. The teaching in England in this matter is almost entirely different from the teaching abroad,—for instance, in Germany. The only country which is following us and getting fairly good results as regards operations for this disease is America. If you have a case of malignant disease, the first consideration is as to whether it is of the intrinsic or extrinsic variety. If it is intrinsic, and the disease is still limited to the interior of the larynx, and there is no glandular infiltration and the general condition of the patient is fairly good, the operation *par excellence* is thyrotomy or laryngo-fissure. It means splitting the thyroid cartilage down the mid-line, and in some cases extending the incision through the cricoid, though that is not generally necessary; then holding the two parts aside, and removing with the knife or scissors the malignant disease on the one side, because it is one side which we are dealing with. Supposing you have a case, as was my lot in the last case I had, where you expect to just split the thyroid and remove the disease with the knife or forceps or scissors, and bring the thyroid together again, and you find on exposing the part that the disease is much more extensive than the laryngoscope gave any indication of. In that case you have to go further and remove one half of the larynx—a much more serious procedure. Therefore if the disease has extended beyond the intrinsic parts, and you have settled to do the operation of thyrotomy, you must extend your operation and remove one half of the larynx. There are other cases where you will find the disease is of the extrinsic variety, and where there is some glandular infiltration in the neck. If that is slight and only on one side, and the disease which is giving rise to it is on only one side of the larynx, and the man is in fairly robust health, you may subject him to removal of one half of the larynx. That is an operation frequently performed abroad, but less often in England. There are cases similar to that which you have seen to-day, and others less extensive where the disease occupies the whole of the interior of the larynx on both sides, where there are glands on both sides of the neck. In some of these cases you may find heroic surgeons performing total laryngectomy, removing the cricoid and all the glands in the neck.

Now we come to consider what are the practical results. In England we almost entirely practise thyrotomy; we make a special point of trying to diagnose these cases early, and if we get them in the early stages and the disease is intrinsic we recommend thyrotomy. I know Butlin and Semon have done a good many of these cases, and there has been a percentage of recoveries of something like 84 per cent. from the operation. Recently Mr. Butlin has had a whole series of such cases without any deaths whatever. So now the operation of thyrotomy should be looked upon as a safe one. It consists very briefly in doing tracheotomy, sticking in a tampon cannula, and ten minutes afterwards splitting the thyroid cartilage accurately in the middle line; the interior of the larynx is then examined, and the diseased area removed with the knife or scissors. The whole of the wound is then closed except its lower part, the tampon cannula removed, and some gauze placed over the wound. In ten days generally the patient can be up and allowed to get about. So if the operation is done properly the results should be excellent, and there should be no deaths from the operation at all. In the old days a tampon cannula was kept in two or three days after the operation to prevent any discharge or blood getting into the wound. But it has been found you can leave out the tampon cannula, and it is safer to do so, because in two or three cases in which the patients succumbed they died from septic pneumonia, which was due to contamination of the spongiopiline which surrounded the tampon cannula. Therefore I recommend thyrotomy invariably in those cases where the disease is of a limited nature. But if the disease is of the extrinsic variety, or there is glandular infiltration, you have to choose between removal of one half or removal of the whole of the larynx. Of course removal of half the larynx is the less severe operation, and in England we very rarely perform total laryngectomy. A few successful cases of this operation have been recorded, but the condition of the patient afterwards is very poor. The only case in which I saw a good result from that operation was one from America, which was

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brought over for the British Medical Association meeting in London. This man had had the whole of the larynx removed on account of malignant disease, and the trachea was stitched to the wound and left open. The man made a very good recovery, and he was living three or four years after the operation. But he was useless for following any occupation, because he had difficulty in breathing, and if he exerted himself he felt faint. In consequence he practically lived in the poor-house over there. Still, he was rid of his disease. The majority of such cases die. I think the mortality from total laryngectomy is about 44 per cent. They die of shock; or a little later they may succumb to pneumonia or bronchitis. Or if the patient has lived long enough he is likely to get recurrence in about three months' time. So if you come across a case of the extrinsic variety in which the disease is very extensive I should advise you to leave it alone and not subject the patient to any extensive operation. You will not be able to get away the whole of the disease. Later on, when the patient gets dyspnœa, it would be well to do tracheotomy, and then the rest of his life will at all events be more comfortable. Therefore, as regards malignant disease, endolaryngeal operations should not be done, and when the disease is intrinsic and limited thyrotomy should be performed. If the disease is more extensive you will consider the advisability of unilateral laryngectomy, but total laryngectomy is an operation which can be done in only a very few cases, and in the cases in which that operation has been done the results have been very poor.

With regard to the after results of removal by thyrotomy, they are distinctly good. A very successful instance of partial laryngectomy was that in one of our well-known magistrates who had malignant disease of the larynx. One half of his larynx was removed some years ago by Hahn, of Berlin. He practised as a magistrate for eight years afterwards, and eventually died of some disease irrespective of malignant trouble. So his may be looked upon as a perfectly successful case; and other cases I know of have lived five, six, seven, and eight years after thyrotomy has been performed for malignant disease.

REMARKS ON SPINA BIFIDA.

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A FEW days ago we had at the clinic a case of spina bifida,* which some of you without doubt will recollect having seen, and to-day we would like to give a little time to the consideration of this congenital malformation. Spina bifida is one of the few malformations which not only offer much scientific interest, but you as physicians will sooner or later be called upon to care for one of these little sufferers, and you should be aware that proper treatment may occasionally be attended with most satisfactory results.

Within the last few years much attention has been given to this affection, for in fact it is relatively frequent in occurrence, and we would put it as representing one sixth of all cases of congenital malformations, and also because the surgical treatment of this condition is improving every day, while successful results are more common. Up to within a few years the statistics of operations performed for the relief of spina bifida were certainly far from encouraging, but the reason for so many failures was due to the uncertainty and lack of an exact knowledge of the pathological anatomy of this malformation; and very naturally as long as the anatomical arrangement remained unexplained not much could be accomplished by surgery, for the proper technique was lacking.

At the present time the pathology of spina bifida is well known, and to von Recklinghausen is certainly due the credit of accomplishing this, for by his teachings many of the old and erroneous ideas were entirely done away with, and new points were established; and by introducing a concise classification he overcame the confusion reigning in the minds of the profession regarding this affection. We would, however, point out that certain points which von Recklinghausen emitted theoretically are still disputed, and therefore require confirmation, and a certain number of questions are not as yet conclusively demonstrated, and cannot be so until further researches are carried on in this direction.

* The history of the case and the result of operation will be found fully recorded in 'Les Archives provinciales de Chirurgie,' April, 1903.

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It is our purpose in this lecture to briefly review the types of spina bifida, and also the diagnosis and treatment of this congenital defect. According to von Recklinghausen's classification, we have certain cases of spina bifida in which no fluid nor tumour protrudes through the vertebral cleft. But as surgically we only have to do with cases of true spina bifida, our remarks will be limited to those conditions requiring operative treatment.

Spina bifida is observed under three different types, to which a fourth may be added, the latter simply representing a combination of two of the three principal types. The forms differ considerably one from the other, and are classed as follows: (1) myelomeningocele; (2) meningocele; (3) myelocystocele; and (4) myelocystomeningocele.

We will first consider myelomeningocele. In this type one is dealing with a sulcus in the skin and a cleft in the vertebræ, with a defective closing of the spine, the membranes of the cord, and a fissure in the cord itself, so that the internal aspect becomes dorsal in position. The three membranes of the cord are wanting on the dorsal aspect, while on the ventral aspect of the malformation, either between the pia and arachnoid, or between the folds of the arachnoid, will be found an accumulation of fluid.

In most cases the tumour is broad, but does not project very much, and is most frequently found in the lumbo-sacral region. But it may also be met with in the cervical and thoracic region of the spinal column. When typical, the malformation is usually only met with in the young fœtus; when encountered in children who only come under observation some time after birth, the cutaneous surface of the tumour has usually undergone an ulcerative process. At the base of the growth the skin presents a rosy tint, and usually gives rise to quite a growth of hair. Next to this we find the so-called zona dermatica, which measures about one centimetre in breadth, and from this zone a second circular zone called the sero-epithelial zone originates, it having a smooth pearly-grey surface and an irregular outline. The apex of the tumour, which is termed the medullo-vasculosa area, is brownish in colour, and composed of spongy tissue, which in aspect is quite similar to granulation tissue. At the upper pole of this area will be seen a fistulous track leading upwards, while at the lower pole a similar sinus will be found running in a downward direction.

The participation which the individual tissues take in the formation of this congenital defect can be explained if one examines a section of the tumour. The latter is simply a pouch, the interior of which is filled with nerve-elements.

The ventral wall of the sac is composed of the dura, while the dorsal wall is, on the contrary, very varied in its structure. The apex of the tumour—that is to say, the area medullo-vasculosa—contains a rich network of blood-vessels, between whose meshes nerve-bundles, groups of ganglion-cells, and neuroglia are found. The pia mater is found most inwardly of all.

At the border of the area medullo-vasculosa, the vascular and nerve-elements decrease in numbers, and in the sero-epithelial zone the wall is only represented by the pia mater, which in turn is covered by a thin layer of pavement epithelium of the skin. The zona dermatica is composed of normal skin.

The dura mater which forms the wall of the sac on its ventral aspect is entirely wanting on its dorsal aspect, for the simple reason that it gradually becomes lost in the subcutaneous connective tissue. The connection between the area medullo-vasculosa and the spinal cord can be demonstrated by making a sagittal section. The sinuses which are situated at the upper and lower poles correspond to the points at which the cord makes its exit, and again enters the central canal.

The layer of skin and the spreading out of the cord correspond to the course followed by the nerves, one of which is given out at the lateral termination of the expansion of the spinal cord, while on the ventral aspect another goes out in the median line. They both gradually converge towards the front, close to each other, and pass under the intervertebral ganglion through the dura. The presence of the lateral nerve-roots is a proof that there is no defect in the dorsal half of the cord, and simply represents a displacement or rather a malposition of the cord.

Regarding the ætiology of myelomeningocele, Förster and Rokitansky supposed that it originated from a ruptured hydromyelus, through which the nerve-elements came in contact with the dorsal wall of the sac; von Recklinghausen, however, conclusively proved that its origin should be

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ascribed to a defective formation of the medullary groove.

Clinically, the tumour is met with somewhat as follows:—A soft, elastic, markedly fluctuating tumour, on the edges of which may be felt the remains of the vertebral arches. Pressure on the tumour will in most instances cause the fontanelle to bulge out. If the fingers be slowly penetrated into the tumour, the dorsal plaque of the vertebral body can be detected. This type of spina bifida is not uncommonly accompanied by other congenital malformations, such as umbilical hernia, exomphalos with intestinal fistula, club-foot, and paralysis of the rectum, bladder, or lower limbs.

By the term meningocele is understood a liquid collection situated between the rachidian meninges, forming a hernia through a defect in the vertebral column. The characteristic feature of this type of spina bifida is that the sac contains no elements of the cord. A typical example of meningocele is rarely met with, and even von Recklinghausen, with the immense material that he had at his disposal, did not have a single example; while Gould, of London, was only able to find two such specimens out of twenty-three preparations of spina bifida.

The participation of each of the membranes of the cord in the formation of the sac is, as yet, an undecided question, but von Recklinghausen is of the opinion that there is a defective development of the dura mater in all types of spina bifida. This teaching appears quite feasible, because it can be readily understood that if a defect in the dura exists, a hydrops can more easily protrude through the membranes of the cord than if the membranes were perfectly developed. On the other hand, it would appear that Hildebrandt's studies go to show that in many cases of meningocele the dura was in perfect condition, and this authority consequently distinguished theoretically several types,—as, for example, when the dura is alone involved, or where both it and the arachnoid compose the walls of the sac; and lastly, those cases in which, the dura being defective, the arachnoid forms the sac.

Meningocele is most frequently met with in the lumbo-sacral region, less often in the cervical region, and only rarely in the thoracic and lumbar regions of the spinal column. The tumour is generally pedunculated, while the skin covering it may be normal or pathologically changed, according to circumstances. To transmitted light the tumour will be found transparent, and cannot be reduced by pressure, although in some cases the fontanelle may be made to bulge.

Other congenital malformations may be present in the same subject. There may be disturbances of the sensibility and movement in the lower half of the body, but this is not frequent. We should never forget the possible presence of the nerves of the cauda equina in the wall of the sac when the spina bifida occupies the sacral region. A rare complication, which may easily lead to a confusion with myelocystocele, is the presence of a very broad cleft in the bone with a resulting prolapse of the cord, which is bent upon itself as it protrudes through the bony gap.

Myelocystocele is a protrusion of the cord through a fissure in the vertebral column, and originates from a local expansion of the central canal. The cord had already become enclosed in the canal, and herein lies the distinction between this type of spina bifida and myelomeningocele, in which case a fluid collection, contained in a sac composed of the arachnoid, projects through a cleft remaining in the medullary canal.

Förster and Rokitansky believed that all types of spina bifida in which the cord is involved owe their origin to a hydromyelus, while Koch regards the latter condition as resulting from a meningocele. It has, however, been demonstrated by von Recklinghausen that only one type of spina bifida originates from hydromyelus. A marked attenuation of the membranes of the cord has caused a confusion with meningocele, and this may explain the fact why so few examples of myelocystocele have been recorded. It is quite probable that many cases that have been considered as meningocele should be classed among myelocystocele. As the latter depends upon a partial expansion of the closed medullary canal, the wall of the sac will be composed of the arachnoid and the pia mater, while the dura is wanting. Interiorly the sac is lined with a layer of cohesive epithelium, which is a continuation of the normal epithelial lining of the central canal, and which also extends to the expanded portion. Between this lies a thin layer of cord substance, as well as ganglion-cells and nerve-fibres. The skin covering the tumour is in the beginning usually normal, but later on ulceration may take place on account of the

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increase of internal pressure as more fluid collects in the sac.

The cleft in the bone is usually limited, and more frequently is situated a little to one side of the median line. This type of spina bifida is more frequently found in the sacral region, but occasionally it occurs in the cervical or thoracic portion of the vertebral column. At times the latter only presents a cleft in its posterior aspect, but in other cases a lateral defect of the spine will be found, such as asymmetry of the vertebral bodies and shortening of the trunk. Frequently the child will also present an extrophy of the bladder, club-foot, or a diastasis of the recti muscles.

Myelocystomeningocele is the type of spina bifida representing a combination of meningocele and myelocystocele. Its cause is ascribed to the fact that fluid collects between the folds of the pia mater and arachnoid, resulting in the formation of a myelocystocele sac, and thus a meningocele has combined with the former condition.

According to the seat of the myelocyst and the manner in which the accumulation of fluid has arisen, we have the three following subdivisions, namely : — (1) Myelocystomeningocele dorsalis, which is a myelocystocele connected with a fluid collected in the dorsal subarachnoidal space. On opening the tumour the interior of the meningocele is first exposed, and at its bottom we find a second cyst, which is the prolapsed spinal cord. (2) The dorsoventralis type. An accumulation of fluid is found in the anterior aspect of the arachnoid, and another between the posterior surface of the vertebral body and the anterior surface of the cord, in close proximity to a myelocyst which is formed by the posterior membranes of the cord, and usually situated close to the dorsal soft structures. The anterior meningocele pushes the cord through the vertebral cleft, which is usually quite broad in these cases. (3) The antero-posterior type. Here the myelocyst is formed by the anterior wall of the cord (thus distinguishing it from the second type), and is surrounded by a meningocele, while the posterior aspect of the cord, which is not attenuated, is connected with the outer covering of the tumour.

When a myelocyst bursts in one of these composite types of spina bifida, and results in a communication with the meningocele, it gives rise to a form of malformation that is often difficult to explain. The structure of the sac in these different types can easily be surmised if one takes into consideration the above description, but the dura will present a dorsal defect corresponding to the cleft in the vertebræ.

The cleft in the vertebral arches in the composite types of spina bifida, especially in the two last mentioned, is quite broad, and can be easily made out by palpation. As in simple myelocystocele, these composite varieties are met with clinically as broad pedunculated tumours, usually situated in the lumbo-sacral region. The skin covering the tumour may or may not be normal. By pressure a considerable bulging of the fontanelle may be produced in some cases. The other malformations which are apt to accompany these types of spina bifida are more especially scoliosis, rarely lordosis; also club-foot, diastasis of the recti muscles, abdominal hernia, etc. Paralysis of the bladder or rectum is rarely present.

The origin of spina bifida consists, according to von Recklinghausen, in a fundamental disturbance of the embryonal tissues, which sometimes only affects the mesodermic layer, but which may also extend to the ecto- and endoderma. The various degrees in the developmental arrest correspond to the various types of spina bifida.

The maximum point of abnormality in development is represented by rhachischisis and myelomeningocele, which consists in a defective closing of the spinal canal, and in a defective formation of the spinal meninges and soft parts covering the dorsal aspect of the spine. If a tumour occurs, as it does in all types of spina bifida excepting the occulta, it is due to a collection of fluid in the subarachnoidal space, which is the result of a chronic inflammatory process occurring in the pia mater and arachnoid.

Von Recklinghausen found that in certain cases of myelocystocele there was an arrest in the longitudinal development of the spinal column, and he explained the origin of the myelocyst from this fact. When the spine remains too short, in consequence of an arrest in its longitudinal development, and the cord continues to develop normally, the latter is obliged to accommodate itself to the limited space given it, and this it accomplishes by becoming bent upon itself. Disturbances in the circulation arise in consequence of this deformity, and a chronic inflammatory process produces a

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circumscribed expansion of the central canal, and the result is a myelocyst.

Virchow's hypothesis may be applicable in other cases. The illustrious pathologist found the spinal cord abnormally long in one case, and he believed that the doubling over of the latter was due to an absolute increase in its length, which destroyed the normal proportions existing between the cord and the spinal column. It is, however, true that a general explanation of the etiology applicable to each and every case of spina bifida is yet to be found. But I cannot refrain from making the assertion that, according to my way of thinking, hereditary syphilis plays an important part as a causative factor of spina bifida, as it does in so many other congenital defects.

In meningocele the defective formation is limited to one or two vertebral arches, and also in the dura. The resulting protrusion of the spinal meninges was believed by Muscatello to be due to a faulty equilibrium between the blood-pressure and that of the cerebro-spinal fluid, and from these disturbances in the circulation a chronic inflammatory process in the pia and arachnoid ensued.

If the arrest in the development is present in both anterior and posterior primordial segments, we then have an umbilical or vesico-rectal extrophy.

Regarding the time at which the disturbing cause acts, it is probable that it takes place with the appearance of the mesenchyme layer, while in myelomeningocele the malformation occurs when the medullary sulcus is still open. The exact nature of the disturbing influence is as yet unknown.

The diagnosis of spina bifida is extremely easy in the majority of cases. The presence of a tumour located along the track of the spinal column at birth, its shape and consistency, as well as the ultimate appearance of pressure symptoms, such as paralysis, are quite characteristic. Exploratory puncture done aseptically will in most cases indicate the character of the tumour, although we can see no necessity for ever resorting to this.

The simple diagnosis of spina bifida is, however, not sufficient in practice, and the surgeon should recall to mind the various anatomical types presented by the affection, because they are so very different from each other, and have the greatest importance relative to the prognosis as well as the selection of proper therapeutic measures. In most cases the differential diagnosis can be made without difficulty, but in some instances it may be surrounded with much obscurity.

Myelomeningocele is by all odds the easiest form to diagnosticate. The typical appearance of the surface of the tumour with its area medullo-vasculosa and epithelio-serosa and dermatica zones, as well as the functional disturbances presented by the patient, such as paralysis of the lower limbs, bladder, or rectum, and lastly the broad cleft present in the vertebral arches, are all characteristic.

On the other hand, meningocele and myelocystocele present a very narrow cleft in the bone; the skin covering the tumour is usually intact, and functional disturbances are infrequent.

The diagnostic differences between meningocele and myelocystocele are not nearly so marked, but there are, nevertheless, a number of distinctive characters which when they exist will allow one to make a probable diagnosis of one or the other type of spina bifida. For example, the usual site of meningocele is the sacral region, and it is quite doubtful if a true type of this form of spina bifida has ever been reported in any other portion of the spinal column.

Myelocystocele may, on the other hand, be present on any portion of the spinal column, but when it is situated low down it is found in the lumbo-sacral region. Now when one observes vascular ramifications in the skin covering the tumour, as well as brown-coloured spots which denote the presence of small subcutaneous hæmorrhages, one is in all probability dealing with a myelocystocele.

If the tumour presents a broad pedicle, it is probable that it is a myelocystocele, but it should be remembered that this condition may also be present in meningocele from the thickening or from an œdematous condition of the subcutaneous tissue or the membranous coverings of the cyst.

A valuable sign for differential diagnosis is the width of the cleft in the spine. In meningocele, simple myelocystocele, and myelocystomeningocele dorsalis the cleft is in most cases too narrow to be made out by palpation. In the two types of meningocele combined with myelocystocele the defect in the spinal column is marked, and can readily be detected by palpation.

From this we may consequently conclude that if a myelomeningocele can be excluded, a spina bifida presenting a cutaneous covering and a broad cleft in the spine is to be considered either a

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myelocystomeningocele ventralis or antero-posterior.

Other congenital malformations and paralysis are rarely met with in cases of meningocele, but a myelocystocele may be accompanied by a number of other defects, such as club-foot, extrophy of the bladder, etc. If in a doubtful case they are absent, it does not necessarily imply that the tumour is not a myelocystocele, but when they are present they positively indicate this type of spina bifida.

Of course an exploratory incision should be resorted to if necessary when there are no contra-indications, and a radical operation may be undertaken if the condition warrants it. Of the older methods of treatment, the only one worthy of mention, and which is still in use to-day, is Morton's injection of tincture of iodine, the formula of which is as follows:—Iodine, 1 part; iodide of potassium, 6 parts; and glycerine, 50 parts.

The sac should be punctured on one side and a portion of the liquid contents drawn off, after which from five to ten cubic centimetres of the iodine compound are injected. The trocar should be of small calibre. It is quite unnecessary to say that the skin covering the sac should be cared for with all detail demanded by aseptic surgery, because if the interior of the sac should be infected a suppurative meningitis would result. The wound made by the trocar should be closed with dry aseptic gauze held in place by collodion. The operation may be repeated four or six times at intervals of ten days.

But with our present knowledge of the anatomy of this congenital affection it would appear as if the proper treatment should be by excision of the sac. This radical interference is not, however, applicable to all cases of this affection, and only certain types can be thus treated; and it is for this very reason that we have reviewed the pathology of spina bifida in order that a clear understanding of the several subdivisions might lead you to make a good differential diagnosis of the condition present in a given case.

The mortality of operative treatment of spina bifida will in the future certainly diminish; as soon as the necessity of an exact differential diagnosis is realised by the profession, and when cases are studied with more care, one will at once be able to eliminate all hopeless cases from those suitable for radical treatment.

Regarding the indications for operation, it may be said that there is nothing as yet decided, but we should never forget to diagnosticate the anatomical type of spina bifida, and to consider carefully the individual case. De Ruyter considers meningocele as the only form of spina bifida suitable for operation, and, as we have pointed out, this form is a simple cyst containing cerebro-spinal fluid communicating with the interior of the spine.

Meningocele should always be treated surgically, even if the tumour is small in the beginning and gives rise to no other disturbances. There is, however, one complication that is prone to occur at any time. I refer to inflammation of the cord. It has been demonstrated that suppurative meningitis may take place even if the skin covering the sac appears perfectly normal. Disturbances of the nervous system are apt to develop gradually and increase in intensity as time goes on, so that they ultimately give rise to neuropathic ulcerative processes. The usual course of events is the extension of the ulceration, the skin becoming eroded, while uncleanliness and friction of the parts from dorsal decubitus add their share in rendering the condition worse.

Necrosis of the skin finally becomes marked, and the cutaneous covering softens, soon giving exit to the fluid contents of the sac; and after the cerebro-spinal fluid has drained away, nervous symptoms appear, the patient rapidly developing unconsciousness, while convulsions supervene. Death soon results.

In operating we should remember that the cavity of the sac may contain nerve-bundles of the cauda equina, which should be carefully handled and returned into the interior of the vertebral column. But should this prove impossible, as, for example, in those cases in which the nerves spread out in a network over the wall of the sac, it is quite proper to remove them along with excision of the sac. The inner lining of the sac containing the network of nerves may also be peeled off and replaced uninjured within the vertebral column. This is, however, a delicate piece of work, because the walls of the sac are very thin and easily lacerated.

Resection of a few branches of the cauda equina does not necessarily result in paralysis, and may be done if the nerve-bundles removed are not of much size.

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Opinions differ as to the advisability of operating in cases of myelomeningocele, and although von Ruyter is decidedly averse to this, Bayer, on the other hand, decidedly favours operation in this form of spina bifida, but personally I am opposed to this treatment.

The technique is as follows :—A circular incision is made and the entire epithelio-serous zone is peeled out of the zona dermatica, while the surrounding structures form two flaps, which are sutured after the area medullo-vasculosa, and all the nerves, are replaced within the vertebral column. The result is that we simply cover the area medullo-vasculosa with the cutaneous flaps, and it becomes adherent to the subcutaneous connective tissue, while the nerve-bundles degenerate in the mass of neoformed cicatricial tissue.

The utter uselessness of operative interference in myelomeningocele is evident. The width of the fissure in the vertebræ, the marked arrest in development of the cord, the secondary changes arising in the unclosed medullary groove, the functional disorders in those organs supplied by the nerves contained within the sac, as well as the section of the cord situated between the lesion, all amply demonstrate why operation is out of the question.

In myelocystocele the inner layer of the wall of the sac is composed of a part of the cord, generally the posterior, which has become intimately adherent to the outer layers, while the cavity of the sac is in communication with the central canal of the cord.

Now if the tumour is removed we naturally are obliged to take with it that part of the cord which enters into the formation of the wall of the sac ; but this does not appear to us to contra-indicate radical treatment in this type of spina bifida, for the reason that the functional activity in the nerve-elements is completely abolished. The cord lining the sac is quite as thin as paper, and it is often with the greatest difficulty that the nerve-elements can be discovered.

By the changes in or the complete destruction of the nerve-elements, the function of the cord is interrupted, and consequently the removal of that part which enters into the structure of the sac can in no manner influence the ultimate condition of the patient; and we certainly believe, in accordance with many other authorities, that this type of spina bifida is quite suitable for operative treatment.

The surgical treatment of the composite forms of myelocystocele differs according to circumstances. According to Muscatello, myelocystomeningocele dorsalis should be subjected to operative interference. The sac formed by the meningocele is removed, and then, if the myelocyst is quite small and can easily be reduced, it should be replaced within the spinal canal ; but if, on the contrary, it is of some size, it had better be removed, because in all probability the nerve-elements contained within it have lost all functional value, and the cyst when placed in the spinal canal would only act as a foreign body.

Operative measures for myelocystomeningocele are contra-indicated on account of the very broad cleft in the vertebræ. The operation, if done, would be the removal of a myelocyst and the emptying of the meningocele, after which the cord should be replaced within the spinal canal. Myelocystomeningocele anterior is not suitable for operation, as it is quite impossible to separate the substance of the cord from the walls of the sac, and to place the former in the rachidian canal.

There are certain conditions which absolutely contra-indicate surgical interference in any case of spina bifida, and we will rapidly mention them :— (1) *The existence of hydrocephalus ;* (2) *a very broad vertebral cleft with a large thin-walled cyst, so that not enough skin can be obtained to make good flaps ;* (3) *the presence of other defects or marked functional disorders, as, for example, paralysis.*

The surgeon should always bear in mind that a hydrocephalus may develop after a successful operation for spina bifida, and for this reason no case should be reported as cured until at least eighteen months have elapsed since the operation.

In operating for spina bifida we have two conditions to fulfil : firstly, the excision of the sac ; and secondly, to prevent a relapse by a complete closing of the vertebral cleft. A relapse in cases in which the bone cleft is very narrow need not be feared, and consequently closing of this cleft is not an absolute necessity.

In a general way the steps of the operation are as follows :—Two elliptical incisions are made in the healthy skin covering the sac, and are then peeled down to the base of the tumour by blunt dissection with the finger. The sac, being thus

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isolated, is incised on one side and its interior explored by the finger in order to ascertain the position of the medullary elements. If the sac forms a simple cyst without any nerve-elements within it, a ligature is placed around the pedicle close to the vertebral cleft and the sac removed. If, on the other hand, medullary tissue is present, as much of the sac is resected as is possible, and the remainder, along with the nerve-elements, is returned into the spinal canal.

To prevent a recurrence it is important to cover the cleft by an osteoplastic operation, after which the skin flaps are brought together with sutures.

WITH MR. D'ARCY POWER

IN THE OUT-PATIENT ROOM AT ST. BARTHOLOMEW'S HOSPITAL.

June 15th, 1903.

GENTLEMEN,—I think that much knowledge is lost in hospitals by omitting to follow the after history of the various cases which come before the surgeon. Cases are admitted into the wards in an acute condition; they are operated upon, and during the convalescence excite great interest. But then, too often, they pass completely out of mind. I propose, therefore, from time to time to show to those attending my class a few cases illustrating the subsequent condition of patients who have been under my care in the hospital for unusual or specially dangerous operations.

(1) *Recovery after a Perforated Duodenal Ulcer.*—The case just coming in is very interesting because it is one of the few cases of perforating duodenal ulcer which have recovered. He is a newspaper manager, and was admitted into St. Bartholomew's Hospital at 3.50 a.m. on July 26th, 1902, suffering from abdominal pain and sickness. He said that he had suffered from similar attacks of pain at various times, but this was more severe, and came on suddenly at 7 p.m. on the previous day. The abdomen was hard, tense, and painful. The temperature on admission was 97·2° F., and the pulse 112. My house surgeon did not think the symptoms particularly urgent, and I did not see the patient until 11 a.m. He then localised his pain over the right iliac region, which was tender and full, the rest of the abdomen moving freely during respiration. I thought he was suffering from an attack of acute appendicitis, which had probably ended in perforation. I therefore opened the abdomen in the right iliac region, and found the appendix normal. Gas issued from the wound as soon as the peritoneum was incised, and there was a gush of alkaline fluid, which did not smell and was clearly bile-stained. The ileum was inflamed in patches, which seemed to correspond to Peyer's patches. I plugged the wound and opened the abdomen by a second incision carried along the middle line. The stomach was found to be normal, but there was a perforation in the duodenum and on its anterior surface, the hole measuring about an eighth of an inch across. It was closed with two layers of Lembert's sutures. Drainage-tubes were inserted at the upper and lower ends of the median incision as well as into the first incision over the vermiform appendix, and the wounds were closed. The patient bore the operation well, his pulse next day being 116, and his temperature 98·8° F. There was much discharge from the drainage-tubes, which were emptied daily by a syringe. The wounds were dressed with boracic fomentations on and after August 4th, and the tubes were replaced by plugs of iodoform gauze on August 5th. The pulse on this day was 96, and the temperature 98·4° F. The patient made a steady recovery, and was sent to the convalescent home at Swanley on September 12th, 1902, the forty-eighth day after the operation. He looks a typically healthy man at present, and there seems no reason why he should not have a long life. The median abdominal wound is perfectly sound throughout; the wound over the region of the appendix is good, but although there is no hernia I do not consider it to be quite as satisfactory as the other.

I thought a note about the feeding would be of interest, and so I asked Sister Kenton to give me her notes of the nursing of the case, and they are as follows:—July 26th, no vomiting after the operation; nothing at all was given by the mouth until after 8 p.m., when he had a drachm of water occasionally. We began to give nutrient enemata at 6 p.m. on the evening of the operation, and they were continued every four hours for the next

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ten days. Each enema consisted of three ounces of peptonised milk, with plasmon, one ounce of essence, and salt half a drachm. Every enema was retained, and the rectum was washed out with plain water twice in every twenty-four hours. A good deal of flatus was passed when the tube was inserted to give the enemata. The patient began to complain of thirst after the first twenty-four hours. This was relieved by repeatedly washing out his mouth with water, and now and again by the injection of a pint or half a pint of water into the rectum. A soap-and-water enema was given on the sixth day, and was repeated on every alternate day afterwards. The action of the bowels was kept regular in this manner throughout, for three grains of calomel given on the eighth day had no result. The patient was dressed twice a day for the first ten days, the three drainage-tubes being sucked dry with a syringe at each dressing. Bile-stained fluid was removed at first, but on the fifth day this gave place to pus. The tubes were removed on the eleventh day, and the wounds were packed lightly with iodoform gauze; most of the stitches were removed on the eleventh day. The wound gaped a little, but the edges were drawn together with a gridiron strapping. The urine was always passed without difficulty and in normal quantities. The patient slept badly for the first five nights, and on each night he had a hypodermic injection of morphia, ♏iv (gr. ¼). During the first two days morphia was injected subcutaneously twice in the twenty-four hours. With regard to the food given by the mouth: during the first twenty-four hours a drachm of water was given occasionally, perhaps an ounce altogether. During the second twenty-four hours a drachm of water occasionally, amounting perhaps to three ounces in all. During the third twenty-four hours a drachm of lemon juice and water occasionally, amounting perhaps to about four ounces altogether. During the fourth twenty-four hours a drachm of Benger's food every two hours, with a drachm of lemon juice and water occasionally. During the fifth twenty-four hours a drachm of Benger's food every twenty-four hours, with a drachm of lemon juice and water as before. During the sixth twenty-four hours a drachm of Benger's food and a drachm of beef essence alternately every hour. During the seventh twenty-four hours half an ounce of Benger's food and half an ounce of beef essence alternately every hour. This feeding was continued for five hours, when the patient had some discomfort and felt sick, so an ounce of whey was given every hour in place of Benger's food and essence. During the eighth twenty-four hours he received two ounces of whey every hour. During the ninth twenty-four hours he had two ounces of peptonised milk every hour, and as a treat he got two ounces of freshly made tea at four o'clock in the afternoon. During the tenth twenty-four hours he had two ounces of peptonised milk every hour and four ounces of essence of tea every two hours. He also had a little baked custard at midday. During the twelfth twenty-four hours he had five ounces of peptonised milk, Benger's food, essence, or tea every two hours, and he again had some custard pudding at midday. During the thirteenth twenty-four hours he had the same amount of fluids and custard, and he had a small piece of thin bread and butter for tea, and some bread and milk for supper. On the fourteenth day he had the same diet, with some jelly and sponge cake added. I have laid stress upon the nursing details in this case because I think the patient owes his life in great measure to the unremitting attention which he received in the ward. Too little thought is devoted by you, as a rule, to the details of the after-treatment when you are students, and you have consequently much to learn when you get into practice. You will notice that I made the mistake common in these cases of supposing a perforated duodenal ulcer to be a case of appendicitis. The cause of the mistake lies in the fact that a few hours after the perforation of a duodenal ulcer the pain and rigidity of the abdominal muscles is localised in the right iliac fossa. This phenomenon, I think, is associated with the fact that the intestinal contents are directed downwards and towards the right side, so that they are first brought into contact with the parietal peritoneum in the right flank. The very ease with which the mistake is made should put every one on his guard, and it should be remembered that whilst the symptoms of acute intestinal perforation are clear enough at first, they tend to become more generalised and less easy to discriminate with every hour which is allowed to elapse before an abdominal section is performed.

(*To be concluded.*)

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THREE LECTURES
ON
RESPIRATORY EXERCISES AND PULMONARY PHYSICS.

LECTURE I.

Delivered at the Medical Graduates' College and Polyclinic.

By HARRY CAMPBELL, M.D., F.R.C.P.,

Physician to the North-West London Hospital, and to the Hospital for Nervous Diseases, Welbeck Street.

GENTLEMEN,—In order to understand the subject of respiratory exercises as a means of treating disease it is needful to know something about what one may term "Pulmonary Physics," a subject, I venture to think, of great importance to the physician. The respiratory apparatus presents many purely physical problems, the understanding of which not only enhances our interest—at least I have found it so—in diseases of the chest, but, as I think, our means of combating them also. For this reason I propose to devote most of my remarks in these lectures to a consideration of the physical problems involved. I shall make the lectures as informal as possible, and I hope no one will hesitate to put questions that may suggest themselves on points which I do not make clear. In this lecture I shall first say a few words as to the shape of the chest and the changes it undergoes in development; I shall then make a brief reference to the pleura and the movements of the lungs; after which I shall pass on to a consideration of the muscles of respiration; and finally I shall deal with the methods by which the chest can be altered in size.

First, as to the shape of the chest. Here I have a series of diagrams illustrating the changes which the chest undergoes in shape from fœtal life onwards. I show you a diagram of the chest at the third month of fœtal life; taking 100 to be the transverse diameter, the sagittal or antero-pos-

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terior diameter at this period is represented by 116, and we say that the thoracic index is 116. Thence onwards the chest flattens sagittally: at the fourth month it is 110; at the seventh month 104; at birth it is 100, the sagittal and transverse diameters being then exactly equal. By the age of five years the chest has flattened so considerably from before backwards that the index is now 85, and the flattening process continues till adult life is reached, when the index is 70. After this the thorax begins to deepen sagittally, and in old age, when the individual is apt to become emphysematous, its shape tends to return to that of the new-born child,—that is to say, to become round. Now you are familiar with the fact that in quadrupeds the thorax is compressed laterally; it is deeper sagittally than transversely, the scapulæ being placed laterally rather than posteriorly, obviously for the purpose of facilitating the backward and forward movement of the fore-limbs in progression. And there is no doubt, I take it, that in the change in shape which the human thorax undergoes during development we have, as in so many other instances, an epitome of man's evolution, or, as we say, of his phylogenesis,—that is to say, man has evolved from a creature which progressed on four legs, from a being whose chest was, like the chests of the dog and the ape, deeper sagittally than transversely. The sagittal flattening of the human chest during development is no doubt connected with man's upright position: the child as it comes into the world is allied to the quadruped, and the chest is round, but as the upright position is assumed the thorax flattens sagittally so as to throw the centre of gravity vertically above the narrow bases of support afforded by the feet.

There is a variety of chest termed the "flat chest," which is not, however, as a matter of fact, a flat chest at all, but actually deeper than the normal, its index being about 80. This was pointed out by Professor Woods Hutchinson in a learned and philosophical course of lectures delivered here to an audience all too small. He showed that the flatness in these cases is only apparent, and is due to a falling forward of the shoulders, which makes the upper part of the chest seem to be flat when viewed from the front; for if you measure these so-called "flat chests" you will find that they are deeper than the normal chest. The same is true also of a large number of phthisical chests; if you compare their diameters you will frequently find that the sagittal diameter is above the normal, and that the thoracic index is about 80 instead of the normal 70. I shall show in due course that this is due to expansion of the thorax from preponderating action of the inspiratory muscles.

I now ask your attention for a moment to the pleura. I would in the first place remind you that this membrane extends upwards about three quarters of an inch beyond the clavicle; that the two pleuræ meet in the middle line in front, from the second to the fourth pair of ribs; also that they do not extend downwards to the extreme limit of the costal arch, one of the reasons for which being that the diaphragm takes its origin from the inner aspect of the ribs forming that arch: in the nipple line the pleura extends to within about one and a half inches of the costal arch, and in the axillary line to within about two and a half inches; posteriorly, near the spine it goes down to the last rib, and even below it; the left pleura is somewhat lower than the right.

The functions of the pleuræ are twofold, the first being to allow the lungs to expand and contract equally in all directions during the respiratory movements: suppose, *e. g.*, the lungs were agglutinated to the diaphragm and to the lower chest walls, it is obvious that a diaphragmatic breath would lead to an expansion of the lower part of the lungs alone, the upper portions remaining wholly unexpanded; and such an arrangement would be most injurious, for it would lead to an undue stretching of the alveoli at the base of the lungs. By means of the pleuræ, however, this difficulty is obviated, for they enable the lungs to expand more or less equally in all directions when a diaphragmatic breath is taken, as anyone can easily test for himself by means of a binaural stethoscope: if you listen with this to the breath-sounds at the pulmonary apices during diaphragmatic breathing you will hear a murmur quite as distinctly there as over the lower parts of the chest, and though I would not like to say that the lungs expand with this kind of breathing as freely in the upper portions as they do in the lower portions, it would be difficult to prove otherwise.

The lungs tend during inspiration to travel forwards and downwards: during a deep inspira-

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tion they cover the heart, and they travel downwards to the lowermost limit of the pleuræ, the movement being greatest in their lower portions. When, therefore, adhesions form here, breathing is more interfered with than when they occur higher up. I have said that the lungs tend to move down to the extreme limits of the pleuræ in a complete inspiration, but although, as we have seen, those limits do not reach as far as the costal arch, one nevertheless gets a resonant note at the extreme limit of the costal arch at the termination of a complete inspiration. I percuss over my liver at the extreme limit of the costal arch in the nipple line, and get, as you hear, a dull note; I now, while continuing to percuss, take a deep inspiration, and you observe how the note varies in tone, how the dulness gets less and less, and how the resonance steadily deepens until it ultimately becomes very pronounced, although all this time I am percussing over the liver beyond the lung—namely, over the edge of the costal arch; now, as I let my breath go, the note gets dull again. For a long time this puzzled me; I could not tell why there should be resonance here over the liver where no lung intervenes. I have little doubt that the explanation lies in the fact that the costal arch is lifted up from the liver during deep inspiration, and thus yields a resonant note.

The second great function of the pleura is to serve as a lymphatic sac; each constitutes a large lymph-space. By the movements of the diaphragm the lymph is pumped from the peritoneal cavity through the diaphragm into the pleuræ, and from the pleural cavities into the lymphatics of the pleura, thence into the bronchial glands, and so into the lymphatic ducts. The movements of the lungs aid the flow of lymph from the pleuræ onwards, and thus facilitate the absorption of lymph and fluid from the pleural sacs; and in this connection it must not be forgotten that the pulmonary lymphatics are provided with valves. Hence the lack of respiratory movement observed in abundant pleural effusions may hinder the absorption of fluid, and sometimes after removing a small quantity of the fluid the rate of absorption is increased, owing, it would seem, to the increased freedom of movement permitted.

In regard to the muscles of respiration, I shall only refer to those which are of practical importance. First of all we have the muscles which dilate the nares. The nares tend to dilate with every inspiration, and in all cases of dyspnœa this dilatation is apt to be exaggerated. Especially is this the case with children; if in them you find marked movement of the nostrils you may be sure that there is urgent dyspnœa, and you may with tolerable certainty diagnose an urgent affection of the chest, such as cardiac failure, or—and especially if the breathing is very rapid—some acute pulmonary affection, such as pneumonia, large pleural effusion, or acute phthisis. A knowledge of this fact is, clinically, very helpful; if a child is breathing quietly, with no movement of the nostrils, you may be perfectly certain, simply by looking at it, that there is no acute trouble of the lungs or heart; if, on the other hand, the alæ nasi are working vigorously, you may be equally certain that some urgent cardiac or pulmonary trouble is present, very rapid breathing pointing to the latter.

The next inspiratory muscles which I will refer to are the abductors of the vocal cords. During inspiration the glottis opens in order to facilitate the ingress of air, and when a deep inspiration is taken it opens very considerably; hence paralysis of the abductors is accompanied by very serious inspiratory dyspnœa: there is great difficulty in getting the breath in, and but little difficulty in getting it out. This condition is happily very rare.

Now we come to the important group of inspiratory muscles situated in the neck. They are—on either side—the sterno-mastoid, the trapezius, and the muscles in the floor of the triangle formed by them and the clavicle—namely, the scalene muscles (which, you may remember, pass from the cervical vertebræ to the upper two ribs), the levator anguli scapulæ, the omo-hyoid stretching across the triangle, and finally, the platysma. All these muscles are, in dyspnœa, thrown into vigorous contraction. As regards the sterno-mastoids, it is important to note that they do not come into inspiratory action until the end of an inspiration, as anyone can prove for himself: if you take a deep inspiration you will find that it is only towards the very end of it that the sterno-mastoids become firm; and this is a point of some practical importance, implying as it does that wherever in dyspnœa you see the sterno-mastoids standing out, the dyspnœa is extreme. With regard to the platysma, for some reason which I cannot explain this muscle is thrown into considerable action in dys-

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pnœa, and in chronic dyspnœics it may get considerably hypertrophied. I do not quite see how it can help inspiration, but there is the fact; you may observe the sheet of muscle, especially in elderly, lean subjects, tightly stretched under the skin, from the lower jaw to the upper part of the chest. Often, too, one can see the omo-hyoid starting out in dyspnœa.

Clinically, the most interesting of all the respiratory muscles, if we except the diaphragm, are, in my opinion, the scalene muscles. In all cases of dyspnœa you find these muscles acting powerfully; if upon placing the hand above the clavicle you feel them working vigorously, you may be sure that there is dyspnœa, and you may measure its extent by the vigour of their contraction; and this is true no matter how quietly the patient may seem on superficial observation to be breathing. All these cervical muscles tend, in chronic dyspnœa, to shorten, and so to fix the chest in a position of expansion. I shall go into this matter in a future lecture, when I shall point out a fact which does not appear to have been generally recognised—namely, that in practically all cases of dyspnœa there is a preponderant action of the inspiratory muscles, one of the results of which in the case of the cervical muscles is that they shorten, and thus tend to fix the chest in a position of inspiration, so that the individual cannot empty his lungs properly, as is well seen in old cases of emphysema, in which the chest is fixed in a position of super-extraordinary inspiration. Physicians have not seriously set themselves to explain this interesting fact; I believe myself that the explanation lies in the fact that the inspiratory muscles shorten from prolonged over-action, just as is seen in the case of certain muscles in talipes.

Next we will take the thoracic muscles of inspiration. Those I would direct your attention to are (on either side) the pectoralis major and minor, the serratus magnus, and a few fibres of the latissimus dorsi. You will remember the latter muscle arises from the crest of the ilium and from some of the spines of the lumbar vertebræ; it also takes origin from the last three ribs at the side, and the fibres springing thence elevate these ribs when the humerus is fixed. The pectorals and the serratus also require that the humerus and scapulæ shall be fixed in order that they may act as inspiratory muscles. This inspiratory action is increased by the elevation of the shoulders, a movement which renders the inspiratory fibres of these muscles more vertical—less horizontal—and thus increases their efficiency as costal elevators. I show you on the screen the portrait of a little girl with very serious heart disease. She has an enormous heart; its transverse diameter must be eleven or twelve inches, and, as may be well imagined, she suffers from urgent dyspnœa. Observe how the shoulders are elevated so that the outer ends of the clavicles are considerably higher than the inner, and how the arms are kept close to the body with the hands pressing down on the bed so as to fix the shoulder girdle. Notice how prominently the lowermost fibres of the pectoralis major stand out in this position, forming an hypertrophied band, the result of prolonged over-action.

Let us now turn to the abdominal muscles. All the anterior abdominal muscles are expiratory—the external obliques, the internal obliques, the transversales, and the recti. Other expiratory muscles are, on either side, the quadratus lumborum which arises from the ilium and sends some of its fibres to the last rib, and certain fibres of the erector spinæ, the outer part of which sends processes to the last five or six ribs, and is thus obviously expiratory; then there is another interesting respiratory muscle, the serratus posticus inferior, which springs from the spines of the lumbar vertebræ and passes upwards and outwards to the last two or three ribs.

In regard to the anterior abdominal muscles, there is one pair which strike me as being especially interesting, and the action of which appears to have been overlooked. I refer to the transversales. These muscles, I may remind you, arise below from the crest of the ilium and the outer third of Poupart's ligament, and above from the last six ribs, while the middle fibres take their origin from the lumbar fascia which is attached to the spine. It is these middle fibres which are of special interest: the upper fibres by their contraction obviously tend to draw together the sides of the costal arch, and the lower fibres to flatten somewhat the anterior abdominal wall, but the contraction of these middle fibres brings the anterior into close contact with the posterior abdominal wall; in other words, the transversales muscles retract the abdomen. By actively retracting the anterior abdominal wall considerable pressure is exerted upon the abdominal viscera,

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and that is done mainly by the transversales, which are thus very interesting muscles.

Under normal conditions the abdominal muscles are moderately contracted—that is to say, in a state of tone,—and they exercise a moderate pressure on the underlying viscera. This is one of the functions of the abdominal muscles—to support and keep in proper position the stomach, liver, and other abdominal viscera. When these muscles are weak and flabby, as happens, *e. g.*, in sedentary people, in women who have worn stays and have thus prevented the abdominal walls from developing properly, in subjects in whom the abdomen has been greatly distended by fluid or fat which has become absorbed,—in all such cases as these the anterior abdominal walls are flaccid, and fail to exert a due amount of pressure upon the underlying viscera, which in consequence tend to move downwards: there is, in fact, a ptosis of the viscera which tends with every added year to get more and more pronounced. Now this downward movement is apt to lead to a dragging upon and stretching of parts—as, for example, the mesentery,—and thus to give rise to a series of unpleasant symptoms such as backache and abdominal discomfort.

Another disadvantage of having flabby, ill-developed abdominal walls is that the accumulation of blood in the splanchnic veins is thereby favoured. Most of you are familiar with the work of Leonard Hill, who has studied the influence of gravitation upon the circulation; he has pointed out that in the upright position the blood tends to gravitate into the most dependent parts of the body, and especially into the great splanchnic veins, which alone, it will be remembered, are capable of holding the entire blood-mass. In order to counteract this a vaso-motor constriction of the splanchnic arteries takes place; I should expect it also of the arteries in the lower extremities, though I do not know that Leonard Hill has studied this point. In some this compensating mechanism is especially efficient, enabling them to stand in the upright position without feeling faint for a longer time than others; in others it is feebly developed, and they are very apt to get tired and faint from standing, owing to the tendency of the blood to accumulate in the splanchnic veins.

Vaso-motor contraction does not, however, constitute the sole factor in compensating the effects of gravity on the circulation: the abdominal walls constitute a second line of defence. When these are firmly contracted the blood is much less apt to rush into the splanchnic area than when they are relaxed, for you will remember that all the veins in the body are highly compressible, being in this respect very different from the arteries, which are comparatively incompressible; by pressing upon the belly it is quite easy to squeeze the blood out of the splanchnic veins, while the blood in the corresponding arteries is but little affected. You therefore can readily understand how, when a person has well-developed abdominal walls, and those walls are always kept firmly contracted upon the splanchnic veins, there is much less tendency for the blood to accumulate in the splanchnic area, and for the individual to become faint, than when those walls are feeble and flaccid. We have here, no doubt, one of the reasons why stays are so popular; doubtless they are worn largely because they are supposed to improve the figure, but they are also undoubtedly worn for "support." If a woman has been in the habit of wearing stays which exert pressure on the abdominal wall as far as the umbilicus, and has thus allowed an artificial support to take the place of Nature's own stays as constituted by the abdominal muscles, especially by the transversales, she becomes dependent upon this artificial support to exert that firm and constant pressure upon the belly which in the upright position tends to retain the abdominal viscera in position and to prevent the blood from gravitating into the splanchnic veins.

The third evil resulting from weak abdominal walls is constipation; nothing, however, need be said on this head here.

I will not take up your time in describing exercises for developing the abdominal muscles, because suitable ones will readily occur to you. I must content myself by emphasising the importance of securing their adequate development.

We now pass on to consider the means by which the chest can be altered in capacity. There are two chief kinds of breathing—(1) the costal and (2) the diaphragmatic. By means of costal breathing the chest is increased in its sagittal and transverse diameters. Let us consider what happens in a complete costal breath: the clavicles are raised to the utmost, and if I now lift them [here the lecturer demonstrated his remarks] as high as I

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can, you will observe that they practically touch my chin; at the same time the ribs move upwards and the sternum is carried forward, and the chest is increased in its sagittal and transverse diameters; then towards the end of complete inspiration the spine moves backwards. (This forward bend of the sternum and backward bend of the spine are well observed in the emphysematous chest.) In the upward movement the ribs are bent and altered in their curves, but I shall not attempt to describe exactly what that alteration is, for the simple reason that I do not know, but I hope soon to get a tracing of a rib in the neutral position as well as in the positions of extreme inspiration and expiration; at present I must be content to point out that the ribs undergo considerable change in shape during deep breathing, and that this is needful for the due expansion of the chest,—that is to say, if the ribs were all perfectly rigid it would be impossible for the chest to expand in the normal way: hence the advantage of having elastic, pliable ribs rather than rigid, unbendable ones.

The complete clavicular breath, in which the clavicles are elevated to the utmost, causes the maximum expansion of the lungs—gives the largest "vital capacity." In my book on respiratory exercises I have termed this mode of breathing the "pan-costal," because, though I think the term "clavicular" more accurately describes it, this latter term has been used by singers in a somewhat different sense.

This is one variety of rib-breathing. I have now to direct your attention to a second variety, in which the clavicles are kept fixed, or only allowed to move slightly at their inner ends, while the ribs are elevated; and inasmuch as it is chiefly the lower ribs which move in this kind of breathing I have called it the "lower costal."

These are the two great varieties of costal breathing, and every one who wishes to understand the subject must learn to execute them himself; certainly, by no other means can he teach them to his patients. Clavicular breathing is easy enough, but it is somewhat more difficult to breathe by the lower costal method.

It is possible to breathe with one side of the chest alone, and it is sometimes advisable to adopt this mode of breathing as a respiratory exercise. In learning it the palm of one hand should be pressed firmly against the corresponding side of the chest, and the body inclined as far as possible towards that side, which should be kept as fixed as possible, while an endeavour is made to expand the opposite side to the utmost.

Table showing the quantity of air which can be breathed by the different methods.

Clavicular		400 cub. in.
Lower costal		210 „
Lower costal supplemented by abdominal		210–270 cub. in.
	According to the degree of diaphragmatic descent.	
Abdominal	Thorax kept fixed in its mean position	110 cub. in.
	Thorax kept fixed in position of costal expiration	170 „
	Thorax kept fixed in lower costal expansion	90 „

This table represents my own breathing capacity when I tested it three or four years ago. The clavicular breath was 400 cubic inches, while the lower costal breath was only 210,—that is to say, very little more than one half. When I take a full clavicular breath you will observe that my epigastrium falls in; this is because the lungs are not, under normal conditions, large enough to fill the chest at its potential maximum capacity, *i. e.* when the clavicles are pulled up to their highest and the diaphragm is lowered to its utmost; consequently when a complete clavicular breath is taken the diaphragm tends to be sucked upwards by the lungs, and the costal arch being carried forward, you can understand why there should be a falling in of the epigastrium. Notice how extreme is my epigastric recession at the present moment; so long as I sustain the complete clavicular breath I am unable by the most forcible contraction of the diaphragm to bulge the epigastrium to any but the very smallest degree; were I emphysematous the case would be different. In advanced emphysema the thoracic cage is often fixed in a position of full clavicular inspiration, and, in spite of this, full diaphragmatic breathing is still possible; indeed, the breathing in such a case may be said

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to be purely diaphragmatic. The explanation is simple—the lungs in hypertrophous emphysema are greatly enlarged, sufficiently so to fill the chest when the ribs are raised and the diaphragm lowered to their respective maxima.

It will thus be seen that there are two forms at least of epigastric recession—an active recession brought about by the contraction of the transversales; a passive recession due to the suction upwards of the diaphragm following on extreme clavicular breathing.

I now show you on the screen the photograph of a man who holds the world's record for weight-lifting. You see that the muscles of his chest are enormously developed; nevertheless his blow ("vital capacity") is only 270. This shows that great muscular development of the chest does not necessarily mean great vital capacity, and it leads me to say that the stories which one reads and hears regarding the extent to which the chest can be altered in capacity are not altogether reliable. One hears of "strong men" being able to modify their chest-girth by thirteen or fifteen inches. Now, 400 is an exceptional blow, and yet in making it I find I only alter my chest-girth by three inches; and such being the case, those who claim to alter their girth by fifteen inches should be able to blow something like 2000! The fact is that the great alteration in thoracic girth which these men are able to effect is mainly brought about by an artful disposition of certain extrinsic muscles of the chest, notably of the latissimi dorsi; the chondro-osseous thorax itself can never, except in the case of freaks, be altered in its girth by more than a few inches.

I may observe, in conclusion, that "vital capacity" depends in large measure upon the degree to which the lungs can be emptied of air. With advancing years the actual air capacity of the lungs tends to increase while the vital capacity as steadily diminishes, the individual becoming less and less capable of emptying his lungs, owing to the increasing immobility of the thoracic cage.

The largest vital capacity I have as yet registered was in the case of a man of thirty (6 ft. 4 in. in height) whose chest measured only thirty-five inches in girth. His blow was 420 cubic inches.

CLINICAL LECTURE ON PELVIC INFLAMMATION.

Delivered at the Medical Graduates' College and Polyclinic.

By G. E. HERMAN, M.B., F.R.C.P., F.R.C.S.

GENTLEMEN,—In my last lecture I showed you how the variations of gonorrhœa are explained by the life-history of the gonococcus.

If gonorrhœa is to be cured, every part of the infected area must be reached. In the case of the cervix uteri into which glands extend deeply it is almost impossible. If the disease reaches the uterus it is impossible that the curette should reach every part of the infected mucous membrane. I mention that because in America some surgeons make it a practice in cases of inflammation of the Fallopian tubes to curette the uterus. They think that otherwise they cannot take away the infection from the uterus. Aran, a distinguished Frenchman, long ago showed that it was not possible with any curette to be sure of reaching every corner of the uterus.

When the gonococci get to the Fallopian tubes, they produce the same effects as they do elsewhere. They penetrate between the epithelial cells, and sometimes set up suppuration, and then the tubes become thickened and more vascular. It is doubtful whether gonorrhœa ever causes a large abscess. Owing to the swelling the fimbriæ approach one another, and the tubes become closed. There are two ways of closure. One is *salpingitic* closure, which results from adhesion of the fimbriæ to one another. The other is *peritonitic* closure, in which the tube is closed by adhesions between the end of the tube and the peritoneum in the neighbourhood. This distinction is not without importance, because if the tube is closed by salpingitic closure it can be removed without leakage of its contents into the abdominal cavity. But if the closure is peritonitic, the tube must be torn in the removal, and pus will escape into the peritoneal cavity.

I said it is doubtful whether the gonococcus ever causes much suppuration in the Fallopian tubes. The gonococcus has been injected into the cellular tissue, and has not provoked suppuration, and many bacteriologists believe that cases of gonorrhœa in which there is much pus

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in the tubes are really instances of mixed infection. When there is suppuration, gonococci are found in the pus-cells, and these cells are said to eat up the gonococci. Another view is that the gonococci eat up the cells, but if so the pus-cells are a diet that suits not the gonococcus, because it soon dies there. When pus is retained in the tubes the gonococcus is not found in them.

Another place in which the gonococcus may cause suppuration is in Bartholin's glands. In that situation it may occur months or years after the first appearance of gonorrhœa. One explanation is that the orifices of the glands are so minute that gonococci do not easily get into them. But a better explanation is that the suppuration is a mixed infection and not due to the gonococcus.

Nœggerath was the first to call attention to the important effects of gonorrhœa in the female. He took a view which, I think, is enormously exaggerated. One can seldom tell what causes this or that by working back from the cases in which the effects are present. If you take a lot of cases of pyosalpinx and inquire how many have had gonorrhœa, that does not give an idea of how often gonorrhœa causes salpingitis. The only way is for the general practitioner who treats men for gonorrhœa to find out how many of them infected their wives when they married. I have no doubt that any man who has long been in practice can point to many cases of men who have had gonorrhœa who have afterwards married, and they and their wives have remained healthy. These after-effects of gonorrhœa take place in only a small fraction of cases. Some years ago I inquired at the Lock Hospital into the number of cases of peritonitis met with in gonorrhœa, and I think the percentage was about five. So long as gonorrhœa in the female does not get to the cervix uteri it is a trifling disease.

Peritonitis may occur from other causes besides gonorrhœa. One is septic infection. The vaginitis with which this begins is sometimes trifling. Bacteriologists say that one of the functions of the cervix uteri is to destroy microbes. It is found that inside a healthy uterus there are no germs. In the lower part of the healthy vagina there are many germs, in the upper part a few, but in the uterus none. Septic infection may spread through the vagina and the uterus to the Fallopian tubes, but I believe there are no symptoms beyond discharge until the peritoneum is reached. Then you get the symptoms of peritonitis — pain, tenderness, and fever, with rigid abdominal walls. In the beginning there are no physical signs, but as soon as enough lymph has been poured out to form a swelling, you can make your diagnosis from the characters of the swelling. The swelling is produced by inflammation passing along the Fallopian tubes to the ovary, both of these organs being swollen and adherent to the neighbouring structures, the bowels and the wall of the pelvis. When you have a tumour so formed I know of no criterion by which you can tell whether the tumour is formed mainly of the ovary or of the Fallopian tube. It is correct, as text-books say, that a dilated Fallopian tube forms a swelling which is retort or sausage shaped, but that is when they are looked at after the abdomen has been opened. But when you examine the patient you cannot identify the sausage or the retort shape. The swelling is to the touch rounded. As both tubes are equally exposed to infection, the disease is usually bilateral. As felt from the rectum it is a convex swelling. The gonococcus does not generally affect the cellular tissue. The swelling does not surround the rectum, and in that differs from the swelling produced by inflammation of the cellular tissue.

We know that gonorrhœal infection and septic infection cause salpingitis and inflammation of the peritoneum and ovary. But it is a matter of doubt whether there is such a thing as salpingitis or peritonitis which is due to cold. Possibly the disease may be due to some germ which would not have affected the mucous membrane of a healthy subject, but which has done so because the vitality of the individual was lowered. I think there is good reason to suppose that an ordinary cold in the head is due to some microbe, and I think salpingitis leading to peritonitis may possibly result from cold, because you occasionally see it in virgins, ending in complete recovery. In these cases it is difficult to know what else caused the disturbance, for there appears to have been no way in which septic germs could have been introduced.

The diagnosis is difficult in the first stage, when the outline of the exudation is not definite. But if the patient is properly nursed and kept at rest in bed, in a few days she will become less tender,

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bimanual examination will be more practicable, and the physical signs can be identified. Pelvic peritonitis is seldom fatal. The importance of tubal inflammation as a cause of pelvic disease was first brought to the notice of the profession by Mr. Lawson Tait. Like most persons who bring novel truth to light, Mr. Lawson Tait rather over-estimated the gravity of these diseases of the tubes. I am sure that it is possible for pelvic peritonitis due to gonorrhœa to get well without leaving any symptoms behind. I recall two such cases. I was called to a patient who had been delivered of a child with an easy labour. Six weeks or so afterwards she began to have a temperature and pain, and when she was examined a lump was found on one side of the uterus behind. Her husband admitted that during his wife's lying-in he had acquired gonorrhœa; and the lady had had a discharge shortly before the development of the fever and the lump. It here seemed certain that pelvic peritonitis was due to gonorrhœa. The patient's illness lasted several weeks, and just when we were beginning to discuss the propriety of operation the patient began to improve. I saw her seven years afterwards, and she had been perfectly well during the whole of that time. There was still a lump in the pelvis, but there was neither pain nor tenderness. I recall another case in which an officer contracted gonorrhœa, had given it to his wife, and she got pelvic peritonitis afterwards. The facts were attested by the doctors who attended both lady and gentleman. The lady came over to England to seek a divorce. I was consulted with a view that my evidence might assist her in her divorce proceedings. There was no doubt about the pelvic peritonitis, shown by fixity of the uterus. I saw the lady a year afterwards, and she was then perfectly well. The uterus was movable, and there was no appreciable swelling around it. These cases make me sure that even when a patient has pelvic peritonitis of gonorrhœal origin it by no means follows that she will never be well unless the parts are removed by operation. Adhesions may be absorbed without causing symptoms.

Sometimes suppurated Fallopian tubes burst and cause fatal peritonitis. In some books you may read of numbers of women walking about with pus tubes in their inside, and in danger of losing their life from bursting of these tubes at any moment. That is, I think, exaggerated. The tubes do not burst in the sense of giving way on account of tension, but owing to an acute ulcerative process. There is a resemblance between inflammation of the Fallopian tubes and inflammation of the appendix. There are appendix cases which cause fatal peritonitis in a short time, and others which cause recurrent attacks of comparatively slight severity. The same thing is true of the Fallopian tubes. Bursting, or rather perforation, is the result of an acute process, and may occur soon after the inflammation is set up. Pyosalpinx, if it perforates, will perforate soon. If a patient has long had a collection of pus in her pelvis from salpingitis, the swelling is then surrounded by a thick, strong wall, and there is no danger of the tube bursting. In such cases the danger is that the patient will continue a chronic invalid; there is small danger to life. She becomes a chronic invalid because she is subject to almost continuous pain, usually described as aching, or burning, in the back, in the lower abdomen, and down the thighs. The pain is so aggravated by exertion that it may incapacitate the patient for taking part in the duties and pleasures of life. The pain is relieved, but not removed, by lying down; it is worse before and during menstruation, because there is then a physiological congestion of the pelvic organs. It is supposed that the ovary may have its capsule so thickened as to lead to difficulty in the discharge of the ova. That is a theory. Continued pain and want of healthy exercise, and of participation in the affairs of life, react upon the nervous system of the patient and bring her into a condition of neurasthenia. As the parts are tender there is generally dyspareunia. This may make the patient anxious about the happiness of her married life. The closure of the tubes naturally causes sterility. It does not always follow that because there are adhesions the tube is therefore closed. I remember one case in which I operated upon a patient for pyosalpinx, and I removed one tube and ovary, but found the other tube and ovary so embedded in the adhesions that I could not identify or remove them. The patient within a year had a baby.

The swelling formed by an inflamed tube and ovary is near the rectum, presses on it, and often causes some rectal catarrh, indicated by discharge of blood and slime from the bowel. If the bowels are so confined that the fæces are hard, the patient

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will get pain when the fæcal lumps are passing the tender part. The swelling may get so large as to press the uterus forward, thus indirectly pressing on the bladder and urethra and causing retention of urine; but that is not common. The pelvic congestion, as a rule, affects the bladder and urethra, and the patient gets pain on micturition and bladder irritation; but this is not one of the most important symptoms. Fallopian tubes are sometimes distended with serum, forming hydrosalpinx, a condition clinically different from pyosalpinx. It is said that hydrosalpinx may become pyosalpinx, because in some cases on one side there is pus and on the other side serous fluid; and that pyosalpinx may become hydrosalpinx, because in pyosalpinx in time the pus-corpuscles may get aggregated together and form cheesy flakes, and the cavity remain filled with serous fluid. I have seldom seen any appearance of these processes. The cases of hydrosalpinx that I have seen resembled ovarian cysts. The walls were almost translucent with adhesions here and there, but the pain that is caused is much more than that of a small ovarian tumour. If you have what feels like a small ovarian tumour, but causes a great deal of pain, the probability is that it is a hydrosalpinx. As to the conditions which lead to the production of this thin, tense swelling instead of the thick and vascular tube containing pus, we know little. If you are interested in hydrosalpinx, read an excellent paper by Dr. Clement White published in the 'Journal of Obstetrics and Gynæcology of the British Empire,' March, 1903. He suggests that it may arise from (1) catarrhal salpingitis, (2) peritonitic closure of tubes, (3) congenital closure of tubes from developmental error.

There is doubt as to whether inflammation ever produces hæmatosalpinx. Those who have examined many Fallopian tubes distended with blood have shown that a large number of these cases are due to tubal pregnancy. Tubal pregnancy is far more common than it was at one time thought to be. This view is supported clinically by the rarity of hæmatosalpinx in unmarried women. Almost every case I have seen has been a married woman. But nevertheless I think there are some cases of hæmatosalpinx that are not due to tubal pregnancy. Observations have been published which show that there is a little sanguineous mucus in the tubes during menstruation. Where the congestion becomes extreme I can imagine that there might be enough blood poured into the tube to distend it. We have evidence of this in some diseases that cause closure of the vagina and closure of the tubes during childhood. In such cases the tubes get filled with blood. Obviously the blood distending the tubes in these cases must come from the tubes themselves. Therefore, if hæmorrhage into the tubes takes place in these cases in which there cannot be any possibility of any sort of pregnancy, I think it may also occur in other cases without pregnancy. In hydrosalpinx, as they often cannot be distinguished from ovarian cysts, there is no doubt as to the necessity of operation.

Suppuration of the Fallopian tube due to septic infection is more extensive than that due to gonorrhœa. The danger of salpingo-oöphoritis and pelvic peritonitis is that it may make the patient a chronic invalid. You can never foretell how long it will be before the patient gets well. If you keep the patient in bed for two months she will probably be free from symptoms, but often they relapse again and again. Just as patients get recurrent typhlitis, so they may get recurrent attacks of pelvic inflammation. The disease can then only be cured by removal of the diseased part which causes the inflammation. A good result follows operation in these cases in proportion to the magnitude of the disease which is removed. If you have a large swelling in the pelvis obviously containing a good deal of pus, there is no doubt about the necessity of operation. In deciding for or against operation, the most difficult cases are those in which there are many nervous symptoms and only a small amount of pelvic inflammation, the patient's incapacity mainly depending on her sensitiveness. If there is no distension of the tube, but only some adhesions, and the patient is neurasthenic, an operation will do no good, but will probably make her worse. If it is a case in which persistent pain is undermining the patient's nervous system, the case is best treated by the removal of the local disease. The treatment without operation consists in giving the patient perfect rest and good nursing. That is as important in the treatment of pelvic peritonitis as in pelvic cellulitis. Poor patients cannot get rest in their own homes, for

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they cannot get rest without good nursing. It is important also that they should have sleep, and therefore, while the pain is acute, opium may be required. But it is bad to give opium for a long period, because it takes away the patient's appetite and confines the bowels. Counter-irritation is of some benefit. If you make the skin sore you diminish the pain in the pelvis. But counter-irritation is only a means of temporarily relieving the pain. It does no good unless combined with rest. In appendicitis the doctrine is now accepted that the time to operate is between the attacks of acute inflammation. The same rule applies to the Fallopian tubes. If you operate during an acute inflammatory attack the infection of the peritoneum may be fatal.

Another important question arises. What is the best operation to do? When there is a large swelling fixed in Douglas's pouch, you can let out the pus and drain the cavity by an incision through the vagina, without opening the peritoneal cavity. If the pus-containing cavity be a tube or a unilocular dermoid, this may cure the patient. If it does not, as the suppurated organ was already adherent, no greater difficulty or danger will be added to a subsequent more radical operation. The swelling may not be so distinctly fixed in Douglas's pouch that you can feel sure of letting out the pus without opening the general peritoneal cavity. If so, a more radical operation must be done.

Mr. Lawson Tait and most of those who immediately followed him were content with removing the diseased parts—the diseased Fallopian tubes or ovaries. Some surgeons in France, Germany, and America have removed the uterus and its appendages by the vagina. It is clear that the patient must be more thoroughly relieved of her troubles if the parts are altogether removed than if some of them are left. If the tubes and ovaries are removed the uterus becomes useless and is liable to disease. The result of the operation much depends on the skill of the person who does it. One who is accustomed to getting out the diseased ovaries and tubes by the abdomen will do it better in that way than if he did it by the vaginal method. An operator practised in the vaginal method will be more successful by it than he would be if he chose the abdominal route. My own experience has been so confined to the abdominal route that I can give little information as to the result of operating by the vaginal route.

Is it wise to remove the ovaries as well as the Fallopian tubes? Mr. Tait said that both ovaries and tubes should be removed. He argued that the cause of inflammation affects both sides equally; often if the ovaries are cut into suppuration is found, and therefore that both tubes and ovaries should be taken away. My own advice is not to remove the ovaries unless there is some evidence that they are diseased, because the removal of the ovaries effects a greater change in the patient than removing the uterus or the Fallopian tubes.

I am sure that Mr. Lawson Tait's enthusiasm led him to exaggerate the frequency with which operations are required for these conditions. Operation is required not as a means of saving life, because pelvic peritonitis is seldom fatal, but as a means of preventing chronic invalidism. Before recommending operation make sure that the condition of the patient is dependent on her local disease, and that it is not a case in which the patient's sensitive nervous system leads those around her to over-estimate the severity of some local pain. The best guide is the physical signs. The larger and more definite the lump, the more clear is the necessity for operation. But when the lump is so small that it is doubtful whether the parts are enlarged at all, whether there is anything more than a few small adhesions, then operation should be undertaken with the greatest hesitation.

The Epileptic Aura.—Spratling classifies and studies the various epileptic auras under four heads: (1) psychic; (2) sensory; (3) motor; (4) irregular. He gives examples of each kind, and reviews extensively the literature on the subject. As to the significance of the various forms of aura, little or nothing is known. The author's experience leads him to believe that the more sudden, severe, and complete the epileptic attack, the less likely is it to be preceded by an aura; while the further the convulsion departs from the classical type, the more common and distinct the aura. Spratling believes that a close analytical study of auras may result in contributing a possible clue to the seat of epilepsy.—*New York Med. Journ.* and *Philadelphia Med. Journ.*, July 25th, 1903.

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TWO LECTURES
ON
SOME PRACTICAL POINTS IN CLIMATOLOGY.

Delivered at the Medical Graduates' College, May 13th, 1903.

By LEONARD WILLIAMS, M.D., M.R.C.P.,

Assistant Physician to the German Hospital;
Editor of the 'Journal of Balneology and Climatology.'

LECTURE I.

GENTLEMEN,—It would be quite impossible in the course of two lectures to deal with such a subject as climatology in the comprehensive manner which its importance demands. I propose, therefore, to confine the first lecture to some of the elements which go to the formation of climate, adopting for this purpose the rather rough but by no means unscientific division into "bracing" and "relaxing," with which every one is familiar; and in the second I shall discuss briefly some of the morbid conditions which are amenable to climatic treatment, and indicate the broad principles which should guide us in making our selections. Although I hope to find time to mention some individual places, this can obviously be done only in the most cursory and perfunctory manner.

The basis of all sound climatology is necessarily sound meteorology. Meteorology deals with weather, and weather in the mass spells climate. Observations with regard to the weather, however accurately conducted, in a place for a month, are valueless; but if the same observations are continued and recorded over a period of years, we have the data by which we are able to estimate the climate of that place. But there are some factors which go to the formation of climate which we can obtain without an appeal to the meteorologist; very important factors, which we can observe for ourselves, which, when determined, will enable us to foreshadow in some measure the facts and figures with which we shall be supplied by the meteorologist. Of these there are but three which need concern us at present: they are exposure, elevation, and subsoil. With regard to exposure, it is obviously a matter of prime importance whether our locality is open to, or hidden from, the sun; whether it is exposed to, or protected from, the cold winds, which, in this country, are the north and east; and whether or not it is within the range of any great modifying influence, such as the sea. These are points which should be carefully ascertained and their probable effects noted, for there are no merits which can outweigh any serious drawbacks in this direction. No combination of climatic virtues can, for example, be held to compensate for an absence of sunshine which is caused by the conformation of the surrounding district, which is therefore persistent and inevitable.

Another important factor is elevation. The higher we go the more rarefied does the atmosphere become,—that is, there is less weight of air, giving rise at high altitudes to a feeling of lightness and buoyancy. Moreover the higher we go the less is the moisture, the purer is the air, and the greater are the daily ranges of temperature. The physiological effects of these conditions are quickened pulse and respiration rate, increased evaporation from skin and lungs, and, after a very short time, increased number of red corpuscles in the blood.

I need scarcely insist upon the influence which the character of the subsoil is liable to exercise on climate. Porous soils, such as chalk, sand, sandstone, and gravel, allow of the free passage of moisture both upwards and downwards, and are credited with being especially healthy. Impermeable soils, such as clay, slate, rock, dolomite, and the like, are supposed to be unhealthy, in that they do not permit of the free passage of water from above downwards. Now, although I would not deny to the character of the subsoil a certain influence on climate, I think there is a very great tendency to over-estimate its importance. Especially is this the case in civilised countries, more particularly in towns, where drainage and other subterranean systems are in vogue; for here the natural tendency of water to stagnate upon or immediately beneath the surface is always nullified by the activity of man. The nature of the subsoil, therefore, though it may, when other things are equal, be profitably considered, should not, as a rule, be set on the same level of importance with the other factors.*

Now before applying to the meteorologist, there

* *Vide* a paper "On the Clay and Gravel Soils of London," by S. D. Chippingdale, M.D., F.R.C.S., 'Journal of Balneology and Climatology,' January, 1902 (vol. vi, p. 14).

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are other points upon which we, as medical climatologists, must always satisfy ourselves with regard to a place which poses as a climatic station or a health resort. Climate is for us either a therapeutic agent or a means to the maintenance of health, and we must be careful that what we may gain in climate we do not lose in other respects. The first question, then, to ask with regard to such a place is, "What is its drainage system, and what its water-supply?" And, having received satisfactory assurances on this, the question of public sanitation, do not be lulled into any false sense of security; but make careful inquiries, from such as are disinterested in the matter, into the question of private sanitation. The condition of the hotels, boarding-houses, and lodging-houses must be above suspicion. Many of the local authorities at these places grant sanitary certificates to houses which have been examined and approved. It is the fashion among certain people to make merry over these certificates by asking what is to prevent something going wrong in the twelve months which elapse between each inspection. There is, of course, nothing to prevent accidents either in this or any other scheme, but the system has at any rate this great merit, that it guarantees that the laying of the drains has been sound, that the method of their ventilation is efficient, that the type of closet is satisfactory, and that the water is not derived from shallow, polluted wells. You will pardon me, gentlemen, for dwelling upon this aspect of the matter; but it is not pleasant, as once unfortunately happened to me, after recommending a family to a health resort, to find that two members of it remained in the local cemetery as the result of typhoid contracted from the drinking-water in their lodgings.

Having now decided these questions, let us make our appeal to the meteorologist. He would, if we would allow him, tell us many things, but we may restrict our inquiries to four points. These are sunshine, rainfall, humidity, and temperature. These four are to a very large extent inter-dependent. Thus rain spells cloud, and cloud means the obscuration of the sun's rays; plenty of sunshine means a relatively high temperature during the day and a fall at sundown, hence considerable variations; a high relative humidity gives rise to equability, that is, absence of extremes of temperature; so that, although we shall now consider these factors in detail, it is important to remember that they cannot really be dissociated.

The effects of the sun in warming the air, especially in moist climates, of warming the earth and thus raising the temperature, are well understood. Recent observations have also established beyond question the bactericide powers of the direct rays, so that we now know that amongst the other blessings which it confers, abundant sunshine gives rise to purity of the atmosphere. Other less understood but much appreciated effects are seen in the uses to which Finsen and others have put the concentrated rays. But when all these have been discounted, there remain the unanalysed and probably unanalysable subjective sensations to which direct sunshine gives rise.

> "Sunbeams pour alike their glorious tide
> To light up worlds or wake an insect's mirth."

And these subjective sensations are merely the result of certain stimuli which, though we cannot define them, we know instinctively to be wholesome and health-giving. The vegetable deprived of sunshine withers and dies. That something of the same effect is produced upon the human being may be believed from the French saying that where the sun enters seldom the doctor enters often. Climatically, then, abundant sunshine is of the very first importance, and no place, in this country at any rate, should be selected even for summer residence which does not afford abundant opportunity for the utilisation of this great remedial element.

And while I am on this subject of sunshine I may as well say what applies to all the meteorological data from which the nature of a climate is deduced, and that is this: do not be satisfied with vague assurances such as "This is a very sunny place," or "It is very warm here in winter," but insist upon facts being laid before you upon which you can form an impartial judgment. A fair average of hours of bright sunshine is, annually, 1500 hours, divided into summer 1000, winter 500. The facts, be it observed, concerning any place which claims to rank as a health resort or climatic station, can always be obtained from the publications of the Royal Meteorological Society.

The rainfall of a place is important to invalids, the majority of whom will not venture out when rain is falling. A moderate amount of rain is not

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only no drawback to a climate, but, in that it brings down suspended impurities, it is one of the agents which help in maintaining the cleanliness of the atmosphere. A moderate rainfall is from 25 to 30 inches annually. The average for England and Wales is 35. To show the importance of ascertaining facts upon this point instead of mere impressions, it is well to remember that there are places with very high rainfalls—so high, indeed, as to be almost incredible when compared with the above-mentioned average. Such are Killarney, 58 inches; Llyndulyn, 104; Seathwaite, 129. No place with a fall of more than 40 inches can make any legitimate claim to rank as a health resort.

In considering the rainfall of a place, it is important to ascertain not only the amount which falls in the year, but also the number of days on which rain is recorded to the amount of 0·01 inch or more. If the total fall is moderate and the number of days is considerable, then it becomes evident that not much falls on individual days. This is the rule in most English stations, but in some of the tropical and subtropical climates—Jamaica, for example—though there is a considerable annual fall, the number of rainy daysis small. This means, of course, that there is a rainy season, which you would naturally direct your invalids to avoid. There are some places even at home which seem to have a tendency to rainy seasons, so that it is as well to inquire not only as to the number of rainy days, but as to the months which contain the majority of these days.

A great French climatologist, on being asked what was the most important factor in climate, replied "Humidity." Not only do I believe this to be correct, but I would go so far as to say that humidity bears the same relation to climate as, according to Demosthenes, gesture bears to oratory. Humidity is a very complex entity. It seems as if it ought to have some connection with the rainfall, but it has not.

I do not, of course, mean to suggest that a fall of rain does not increase the atmospheric humidity on a day which up to that time had been dry. Every one is familiar with the softness and freshness which rain under such circumstances produces. What I do mean is that the humidity of a place as observed over long periods of time has no ascertainable relation to the rainfall of that place as observed similarly; that, in fact, there are many places which have a high humidity and a low rainfall, and others which have a high rainfall and a low humidity.

Humidity is of two kinds, absolute and relative, and these two kinds have no ascertainable relation to one another. Absolute humidity is the actual number of grains of vapour in a cubic foot of air. Relative humidity is the percentage of moisture in the atmosphere, saturation being taken as 100. To show the difficulty of stating one in terms of the other, let me quote the following:—"If the inspired air contains 3 grs. of aqueous vapour per cubic foot at 50° F., it has a capacity for an additional 15·7 grs. at 98° F., and its relative humidity is 74 per cent. If the air is inspired at 42° F. with the same amount of moisture, viz. 3 grs., it still has the same capacity, but its relative humidity is now 98 per cent."*

When humidity without any qualifying adjective is mentioned, relative humidity is usually meant,—that is, the percentage of moisture in the air. Anything over 80 is considered high; anything approaching 60 is deemed low. This is assuming the temperature of the air in both instances to be 55° F. No comparison is possible between the relative humidity of different places unless the external temperature at the time of observation is the same in all cases. Now a high relative humidity is productive of equability of temperature. The moisture in the air plays the part of a wet compress, and hinders the passage of heat from the earth. When the temperature falls, moisture is deposited from the air; hence there is a tendency to the formation of mist or fog. Where the relative humidity is low, there are very considerable variations of temperature. It is warm in the day and cold by night, and there is much less tendency to the formation of fog.

One word on this question of the formation of fog. When the temperature falls suddenly, the moisture contained in the air is deposited. This may be seen exemplified on the outside of any glass in which iced fluid is contained. The suddenly lowered temperature in the immediate vicinity of the glass causes a deposit of moisture on the outside of the glass. Dew on the grass is the result of the same cause. Fogs are brought about in a manner precisely similar, but to the

* Dr. C. W. Buckley, 'Journal of Balneology and Climatology,' April, 1903.

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formation of fog or mist, a still atmosphere is essential. Now London fogs and the fogs of other great cities are injurious not because of their moisture, but because that moisture is deposited upon the particles of dust and other irritating matters which are floating in the air, and the weight of the deposited water brings these particles down to the ordinary levels, so that they are inhaled into and irritate our air-passages. By the stillness of the air, and by bringing down the irritating particles which, under ordinary circumstances, float above us, fogs intensify the impurity of the air. It is this, and not their moisture, which constitutes their danger to bronchitics and other lung invalids.

On the subject of temperature not much remains to be said. It is influenced by so many considerations, principally by exposure and humidity, that, given these, the temperature may almost be deduced. The matter is one of simple thermometric measurement, and the recorded monthly means will give us nearly all we require. More important, however, than the monthly means, when they are to be obtained, are the means of monthly minima. The maxima are interesting and important, but from the point of view of the medical climatologist it is the minima which yield the most useful information. The two together will tell us not only to what level the ordinary temperature attains, but what its range is. In nearly all cases of chronic invalidism, extremes of temperature are undesirable.

The climate of these islands is characterised by extremes of temperature; and however well we may recognise that such conditions conduce to the physical hardiness and to the mental and moral characteristics of our race, as I believe they do, we must also recognise that what may be for the benefit of man in the mass is by no means necessarily so for the individual, especially if that individual be very young, very old, or very delicate. Those, therefore, who can be placed in these categories should have the matter of temperature studied for them, so that they can obtain as much warmth in the depth of winter and as much freshness in the height of summer as circumstances will permit. A judicious selection of climates at the various seasons is one of the most powerful therapeutic agents at our disposal in dealing with such cases.

Now let me endeavour to group these various factors, and state them in terms of the "relaxing" and "bracing" which are so dearly beloved of the laity. To the term "relaxing" I would prefer "sedative," as being more correct, and, to medical ears, more expressive.

In a general way, then, and remembering always the existence of many and powerful modifying influences, we may say that, other things being equal, a sheltered situation will have a sedative, and an exposed situation a bracing effect; that a low-lying place is relaxing while an elevated place is stimulating; that a humid and therefore equable climate is sedative, and one which is dry and therefore liable to extremes is exciting. The type of the one may be taken as a low-lying inland valley, thoroughly sheltered from the cold winds, with a high relative humidity and its attendant equability and liability to mists; and of the other, a place with an elevation of about 1000 feet, unprotected by neighbouring hills, with a low relative humidity and its attendant rarefaction of the air, absence of mist, and sudden and considerable variations of temperature. Although it is not my intention to give you anything in the nature of a list of places affording particular climates, an instance or two is always helpful, so I select as good examples of bracing climates Buxton, Ilkley, Strathpeffer, and the east coast watering-places in this country, and Davos and Les Avants in Switzerland; of sedative climates in this country, Cheltenham and the watering-places on the south-west coast; and abroad, Arcachon, the island of Madeira, and the Canary Islands.

Now the physiological effect of a bracing climate, speaking broadly, is that the human machine is driven at high pressure, metabolism is increased, appetite is heightened, and mental and physical energy are augmented. A sedative climate produces the opposite effect. Here the "wheels of being" are slow: metabolism is diminished, the desire and necessity for food are decreased, while physical and mental energy are depressed. The therapeutic effect of the one is comparable to that of ammoniated quinine; of the other, to that of bromide of potassium. And believe me, gentlemen, in prescribing the climates you will have every bit as much cause to regret an error as you would have in prescribing the drugs.

Indeed, in the case of climates the matter is infinitely worse, because you cannot, as in the case

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of drugs, cover your mistake by a reversal of your policy at the next visit. If you advise a particular place and your patient accepts your advice, you have burnt your boats; for do not forget that for any want of success, you may expect your full measure of reproach and discredit.

INTERNATIONAL CLINICS. Vol. iii, twelfth series, 1902, and vol. iv, twelfth series, 1903. (London, J. B. Lippincott Company. Price 10*s.* 6*d.* per vol.)

These volumes are occupied with the clinical lectures of teachers from all parts of the world, on all the general and special branches of medicine. They have deservedly secured a prominent place in current medical literature, and should prove of great value to the general practitioner who desires to keep abreast of the times and has not the opportunity of visiting the great medical centres. One of the lectures which strikes us as specially interesting and valuable is by Professor Dieulafoy, of Paris. In it he discusses first the question as to whether a pleurisy *a frigore* ever occurs, and the various experiments and tests made to demonstrate the tuberculous nature of cases so described. He then proceeds to describe fully the value of cyto-diagnosis in distinguishing between (1) the pleurisy of venous congestion, as in heart disease; (2) acute infective pleurisy, as in typhoid fever; and (3) tuberculous pleurisy. This examination of the cellular elements is not one which can be made only by experts, but is easily carried out by the practitioner who can use his microscope and the ordinary staining fluids. Finally he describes the clinical course, dangers, prognosis, and treatment of tuberculous pleurisy. The indications for surgical intervention in the treatment of dilatation of the stomach are discussed in two interesting papers by Dr. G. M. Debove, of Paris, and Professor Antonio Cardarelli, of Naples. These two physicians lay down with great minuteness the methods of diagnosing the exact conditions present, and the indications where medical treatment can go no further. It may almost be called the dying song of the poor physician, who will soon find himself in a pauper's grave, while the surgeon whom he has taught will be banking his operation fees with the prospect of a baronetcy, or even a peerage. Another paper on a similar subject is that by Mr. Moynihan on the surgical treatment of hæmatemesis from gastric ulcer. These subjects show how well up to date the clinical material of the lectures is. Special articles are devoted to Pavloff's researches on the functions of the digestive glands, to the theory of inflammation, and to the blood in health and disease. The busy practitioner will find some lighter reading on the training of his medical son in the latest scientific methods of diagnosis, and on how to avoid a suit for malpraxis in connection with fracture and dislocation cases. He will also have an opportunity of reading in these volumes the biographies of two eminent American practitioners, who are stated to be still alive, although the accompanying photograph of one of them has a distinctly post-mortem appearance. These self-sacrificing Americans are ready not only to contribute papers, but to contribute themselves in the interests of medical literature. So far as we are aware, no English physician of standing has allowed himself to be pilloried in this manner.

Dissemination of Typhoid Fever by the Means of Butter.—Carl Bruck, by experimental researches, shows that when butter is made from milk infected with typhoid bacilli, the bacilli pass from the milk into the cream and thence into the butter, where their presence may be demonstrated. It is thus apparent that butter is one of the chief factors in the indirect transmission of typhoid fever. The experiments were divided into three series. In the first series, pure cultures of typhoid bacilli were added to the milk from which the butter was made. In the second series, the milk cans were washed out with water which had been infected with typhoid bacilli. They were then filled with the uninfected milk from which the butter was to be made. After a short time the cream was centrifugalised from this milk and butter made therefrom. The third series were identical with the second, except that the water used to wash out the cans was infected with the stools of a typhoid-fever patient, in which stools typhoid bacilli had been previously found. In every series the result was positive, typhoid bacilli being isolated from the butter.—*Medical Record*, July 25th, 1903.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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ON THE DIAGNOSIS OF CARIES OF THE SPINE.*

By JAMES BERRY, B.S., F.R.C.S.,

Surgeon and Lecturer on Surgery, Royal Free Hospital.

WHEN your Committee did me the honour of asking me to read a paper before the North London District of the Metropolitan Counties Branch of the British Medical Association, several subjects at once suggested themselves. But it seemed to me that I should best fulfil the task that devolved upon me if I chose a subject with which we were all more or less familiar, and which presented sufficient difficulty and sufficient points of interest to provoke discussion, and to lead, as I hope this paper will, to a lively debate. All of us, whether surgeons, physicians, or general practitioners, frequently come across cases of caries of the spine, and I do not suppose that there is any one here who will not admit that the diagnosis is often a matter of great difficulty. Speaking for myself, at any rate, I am conscious of having many times overlooked early caries of the spine, or made some other error in diagnosis.

I propose, therefore, to speak of difficulties in diagnosis, and of the best means of avoiding them. The cases that I shall relate in illustration of my paper are all cases that have come under my own observation.

The frequency with which caries of the spine is met with in ordinary hospital practice is only too well known. Tuberculous caries of the spine is a more common affection than that of any other single joint. According to the statistics of the Boston Children's Hospital (quoted by Whitman in his recent excellent book on Orthopædic Sur-

* Delivered at the Royal Free Hospital on February 11th, 1902, at a meeting of the North London District Metropolitan Counties Branch of the British Medical Association.

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gery), among 3720 cases treated at that institution during a period of twenty-four years, rather more than half were cases of tuberculosis of the spine; rather less than half were cases of tuberculosis of the hip, knee, ankle, shoulder, elbow, and wrist combined.

In discussing the diagnosis of caries of the spine, I think we shall do well to bear in mind that the numerous signs and symptoms of the various forms of this disease may conveniently be divided into three main groups, namely:

(i) Those referable to the bony vertebral column itself;

(ii) Those due to the presence of abscess; and

(iii) Those due to involvement of the spinal cord.

In approaching a case of supposed caries of the spine it is well to bear these three groups in mind, and, from the point of view of diagnosis, to investigate each of them more or less separately.

Group I. Signs and symptoms referable to the bony vertebral column.—The normal vertebral column has certain well-marked antero-posterior curves, but is otherwise straight. It is essentially flexible, its flexibility varying as a rule inversely with the age of the patient, since with advancing age the bones and ligaments become harder and more rigid. The movements of a normal spine are, moreover, painless.

We have, then, to consider shape, flexibility, and the existence or otherwise of pain on movement.

Shape.—Of the well-known angular curvature of tuberculous caries of the spine but little need be said, since when it is present there is usually but little difficulty in diagnosis. It can be produced only by destruction of the anterior part (the bodies) of the vertebræ, and (apart from injury) the only other disease that destroys the vertebral bodies and produces angular deformity is malignant disease.

Both sarcoma and carcinoma may produce an angular curvature that is indistinguishable as regards its shape from a typical case of tuberculous caries. But many vertebræ are seldom involved in malignant disease; the curvature that is produced presents usually a well-marked angle or point, and we do not see in this disease the rounded prominence that is so common in connection with tuberculous caries, and which is illustrated by so many of the cases before you.

As an illustrative case of secondary carcinoma I may mention the following:

Epithelioma of œsophagus; development of angular curvature in dorso-lumbar region from secondary growth.—A coachman, æt. 46, was admitted on January 14th, 1884, into St. Bartholomew's Hospital, under Sir Dyce Duckworth, to whom I was at that time house physician. Symptoms of malignant stricture of the upper part of the œsophagus had been present for some six weeks.

A fortnight after admission he began to complain of a good deal of pain across the loins, and there was some tenderness about the lumbar spine. The pain rapidly became much more severe, and the patient was soon quite unable to sit up in bed. On February 20th a well-marked angular curve was noticeable in the spine, the last dorsal vertebra being prominent. Any attempt at movement caused much pain. The patient gradually became worse, and died on March 22nd.

At the post-mortem it was found that the upper lumbar and twelfth dorsal vertebræ had been very extensively destroyed by new growth, secondary to the epithelioma of the œsophagus.

Erosion of the vertebræ, especially the dorsal vertebræ, is often produced by aneurysm, but this form of erosion does not affect the intervertebral discs, and does not usually extend deeply into the vertebræ. Consequently aneurysm does not cause any visible external deformity or angular curve.

Here is a museum specimen which is a typical example of the erosion caused by aneurysm. You will see that there is no spinal curvature. Angular curvature due to tuberculous caries is naturally most marked in the dorsal region, which is convex posteriorly.

The cervical and lumbar regions are concave posteriorly, and are, moreover, covered by muscle and other soft structures. In these regions we do not expect to find so marked a projection. But a diminution or flattening of the normal concavity is equally characteristic of absorption of the vertebral bodies. At a later stage the disappearance of the normal cervical or lumbar hollow gives place to an actual prominence.

The rounded prominence of advanced upper dorsal caries at first sight presents some similarity to the hump of so-called lateral curvature. The latter is, however, easily distinguished by the great amount of rotation of the vertebræ, which causes

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the ribs to project backwards. In lateral curvature it is the ribs that form the prominence; in caries it is the vertebræ themselves.

It must be remembered, however, that a certain amount of lateral curvature and even of rotation occurs, although very rarely, in true caries, especially when this affects the upper dorsal region. A case of slight deformity without acute symptoms sometimes renders the diagnosis between lateral curvature and true caries very difficult. I will cite two cases.

A boy æt. about 15 was brought to me some years ago on account of some irregularity of the upper dorsal spine. There was a distinct lateral curvature, and one shoulder was higher than the other. There was practically no angular curvature, and the amount of pain was trivial. There was no local tenderness. I was in considerable doubt as to the diagnosis, but inclined towards simple lateral curvature (scoliosis). However, a few months later distinctive signs of caries supervened, and paraplegia set in. He drifted off to another hospital, where laminectomy was performed, and at the present time he is dying of spinal sclerosis.

The second case was that of a boy æt. 4½ years, whom I saw in July, 1901. He had a distinct but slight prominence of the second dorsal vertebra. There was absolutely no pain, and the child was running about freely. A doubtful diagnosis of lateral curvature had previously been made, but the subsequent occurrence of paralytic symptoms proved conclusively that the case was one of caries.

In infants and young children it is not uncommon to meet with a backward prominence of the whole spine, which may easily be mistaken for caries. Rickets and general debility are common causes of this condition. It can usually be easily distinguished from caries by the absence of any definite local prominence, and by the fact that the curvature disappears when the child is lifted up by the shoulders.

In the absence, then, of a definite and characteristic angular curvature, the diagnosis of caries of the spine is made, as far as shape is concerned, by the straightness of a portion of spine that should be curved, the obliteration of the normal cervical or lumbar hollow, or by the existence of a single prominent vertebra. The latter condition is one of great importance in the diagnosis. It may be well to mention that the very slight degree of curvature which results in the projection of a single vertebra is generally best detected by looking sideways at the patient's back. A slight projection, not visible when the back is examined in the ordinary way, becomes easily perceptible if the patient is put on his side and the profile view of the spine is inspected.

Flexibility.—As I have said, the spine is normally a flexible structure. But the flexibility varies considerably with the age of the patient. It is mainly in the case of children and young persons that stiffness of the spine—that is, loss of flexibility—is of value as a sign of caries. Middle-aged and elderly people, in whom the bones are harder, and whose cartilages and ligaments tend to become ossified and rigid, naturally have spines that are not as flexible as they once were. Rheumatoid arthritis, a common thing in later life, does much to stiffen the spine by means of the coalescence of the osteophytic bony plates that we find at the margins of the vertebral bodies. I show you here a specimen of vertebræ from a typical case of rheumatoid arthritis, illustrating this condition.

Making due allowance, then, for the stiffness produced by advancing age and by rheumatoid arthritis, a spine should be flexible. It is well in testing the flexibility of a spine to adopt a uniform system of examination. If the patient is able to stand up he should be told to keep the knees straight and then to bend forwards, backwards, and laterally, and finally to twist his head and shoulders round while the pelvis is firmly grasped by someone else. Or, if the patient be seated, he is asked to bend forwards until the head is between the knees. In this position the spine should form a uniform gentle curve from sacrum to occiput. Then a hand should be placed firmly against the sacrum, and the patient told to bend his head backwards until it touches the couch on which he lies.

It need hardly be said that in acute cases such movements cannot be performed at all. In the more chronic cases even a very slight amount of stiffness is in this way easily detected.

We are all thoroughly familiar with restriction of movement as a sign of disease of such joints as the hip, knee, and shoulder, but I think we are, perhaps, a little apt to forget that the spine is a row of joints, and that disease of any of these joints

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causes spasm of muscles, tending to fix the joint in exactly the same way as spasm of the muscles round the hip or knee causes fixity of those joints.

Pain.—I come next to the subject of pain, and especially pain on movement. This is such an obvious and well-known symptom of most cases of spinal caries that I need not dwell upon it. I would rather dwell on the fact that pain may be completely absent in an otherwise typical case. I am inclined to think that it is this very absence of pain that is, in so many cases, responsible for errors in diagnosis.

There seems to be in the minds of many practitioners a great reluctance to diagnose caries of the spine unless pain is a well-marked feature. I have in my mind at the present time the case of a little boy whom I have seen from time to time in the last eighteen months, the same case that I referred to when speaking of the diagnosis between lateral and angular curvature. That boy has a perfectly distinct, although slight deformity of the spine, and he has had partial paraplegia. There is not the slightest doubt of the nature of his malady. And yet there is a certain worthy doctor who occasionally sees him and expresses the opinion that there is really nothing the matter with the boy, and that keeping him lying on his back is merely unnecessary cruelty on the part of myself and the other surgeons who have seen him.

The spine is no exception to the rule that any joint affected with tubercle, even very extensively affected, may be quite painless. Cases of spinal caries without pain are very apt to be overlooked, and the disease is in such cases often allowed to progress to an advanced stage before proper treatment is undertaken.

I must now say a few words about the converse group of cases—those in which pain is the only prominent symptom. Not pain in the back. Pain in the front of the chest, pain in the front of the abdomen, pain down the thighs, pain at the back of the head, pain almost anywhere except in the back. These are the cases in which caries of the spine is apt to be overlooked.

Here is the case of a boy who was taken to a doctor by his mother for a pain in the pit of his stomach, and he was treated for a stomach ailment. No examination of the back was made. It is only fair to the doctor, however, to say that at the next visit a few days later an examination of the back was made, and dorso-lumbar caries was discovered.

I will relate one other case bearing on this point. A few weeks ago I was consulted by a lady æt. 30, who for no less than four years had complained of a well-nigh continual pain in the region of the sternum.

She had consulted numerous surgeons and physicians both in England and abroad. Some had said she was hysterical, others said she had lateral curvature, others that there was nothing the matter with her. She had tried baths and exercises and rubbing, and all sorts of medicine, but the pain remained and she was rarely free from it. Finally she came under the care of another doctor, who noticed a new feature of the case; and when he brought her to see me, having himself already made the diagnosis, the case was quite clearly one of cervical caries. Skiagrams were taken, and they show very clearly not only well-marked caries in the neighbourhood of the fourth cervical vertebra, but very considerable disease of the third lumbar vertebra as well. This is an example of quickly progressive caries without sufficient external deformity to attract attention. This lady's pain was at once relieved when she took to a continuous recumbent position.

Pain in the hips and lower limbs is of course a very frequent sign of caries of the lower lumbar and sacral spine. Only too often does this lead to the erroneous diagnosis of hip disease. Caries of the lowest parts of the spine does not readily cause deformity of the back. It does, however, cause flexion of the hip, pain in the hip, and lameness. It is the presence of one or more of these symptoms that causes so many spinal patients to be sent to the Alexandra Hospital for Hip Disease, with an erroneous diagnosis of hip disease. So large is the number of cases of this kind that have come under my notice during the fifteen years that I have been surgeon to that institution, and so numerous also are the cases in which hip disease co-exists with spinal disease, that I have now laid down a definite rule that I will not consider the examination of any case of hip disease has been completed until the spine also has been examined, and the question of the existence of spinal caries fully considered.

The cases of spinal caries that are likely to be mistaken for hip disease fall into three classes :

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(*a*) The early cases, when the chief symptoms are pain in the hip and thigh, flexion of the hip, and lameness.

(*b*) Cases with abscess, when the suppuration has extended into the upper part of the thigh under Poupart's ligament or through the sacro-sciatic notch. In these cases the suppuration may so surround the hip-joint, and cause so much fixity of it, that the resemblance to hip disease is very close indeed. I frequently find myself unable at a first examination to say definitely whether a child is suffering from hip disease or lumbo-sacral caries, or both.

Here is a case of a child who came to the Alexandra Hip Hospital many years ago on account of supposed hip disease. The history was that she had been in another hospital under another surgeon for pain in the hip and an abscess in the groin. At that time the doctrine that cases of hip disease with suppuration ought to be treated by excision of the hip was taught much more extensively than it is at present. Anyhow this child's hip had been excised, and I took the trouble to look up the early notes of the case, which left no doubt at all in my mind that excision of a healthy hip had been performed. At that time, and for long after I first saw the case, there were no signs of spinal caries. But that such was the true nature of the case became only too plain at a later period, when the characteristic angular curvature, which you see here, set in.

(*c*). The third group of spinal cases which simulate hip disease are advanced ones, where there are numerous sinuses about the hip and thigh, and where the thigh itself is flexed upon the abdomen.

I have even heard of a case of this kind in which an amputation at the hip-joint was actually performed under the mistaken notion that the surgeon was dealing with a case of hip disease.

I have devoted most of the time at my disposal to the consideration of the first of the three groups of symptoms to which I called your attention at the beginning of my paper. I come now to

Group II. Signs and symptoms due to the presence of abscess.—At the outset it may be well to call your attention to the fact that in most cases the so-called abscess of spinal caries is really a collection of broken-down tuberculous matter. Some have objected altogether to the use of the term abscess, but this seems to me to be going too far. Certainly the term abscess is a convenient one, and there can be no harm in its employment provided that we clearly bear in mind that we are dealing, not with an ordinary abscess of mixed infection, but with one in which one organism only—the tubercle bacillus—plays as a rule the important part. It is for this reason that we ought, in dealing with a tuberculous abscess of the spine (or any other joint), to be just as careful with our asepsis as we should be if we were doing an abdominal section or any other aseptic operation. And yet how often do we see spinal abscesses opened and drained with a lamentable lack of ordinary antiseptic precautions, and how great is the evil that ensues when a mixed infection is allowed to occur!

The abscess of spinal caries originates in the vertebral body or disc, and thence makes its way externally. It usually makes its way to the front and sides of the vertebral body, and in rare cases passes backwards into the vertebral canal, where it comes in contact with the spinal cord. Abscess in connection with caries of the cervical spine is especially apt to be mistaken for the common suppuration due to tuberculous glands. When it appears in the form of a post-pharyngeal abscess it may easily be mistaken for gumma or sarcoma. It is well to remember that the front of the bodies of the upper cervical vertebræ are most accessible by means of a finger introduced into the throat.

An abscess in the dorsal region may make its way through between the transverse processes or ribs and project under the skin of the back. I have known such cases mistaken both for sarcoma and for fatty tumour. Sometimes an abscess coming through in this way is derived from an empyema rather than from the spine.

It is well to remember that empyema itself may be set up by an abscess formed in connection with dorsal caries. Any one looking at this specimen of a tuberculous abscess, which has pushed the pleura forwards and outwards, will readily understand how easily it might have extended to the pleura. I have seen a child die of suppurative pericarditis caused in a similar manner by extension from the dorsal spine.

Abscess connected with dorsal caries can seldom be diagnosed unless the abscess extends to the back or down into the abdomen, or unless the patient be examined with X rays. I show you

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here a skiagram of a case of dorsal caries, in which it appears that there is a tuberculous abscess.

Abscesses at the back of the abdomen connected with caries of the dorsal or lumbar region are most likely to be mistaken for abscesses due to disease of an abdominal viscus such as the kidney, or else for new growths springing from the vertebræ or neighbouring parts.

Abscesses connected with caries of the sacrum, or any spinal abscess descending into the pelvis, are likely to be confused with numerous other suppurative diseases. If the caries is in the sacrum there may be little or no curvature of the spine, and it may be extremely difficult or impossible to ascertain the source of the suppuration. Abscesses passing out through the sacro-sciatic foramen or down into the ischio-rectal region are not unlikely to have a spinal origin.

Abscesses passing to the neighbourhood of the hip are especially liable to be mistaken for hip disease. Spinal disease simulating sciatica is of course very well known. The chief source of error in the diagnosis of spinal abscesses arises, I think, from the fact that in their earlier stages at least such abscesses are usually painless and devoid of tenderness.

Before concluding this part of my subject I will refer to one more case—that of a man who was recently in my wards at this hospital. He had had a firm, painless swelling in his right lumbar region, which had been explored by operation at another hospital, and was thought to be a sarcoma. It did not, however, run the usual course of a sarcoma, and some months later he was sent up to me for further operation. At this time there was a slight —a very slight—prominence of the last dorsal vertebra, and, to make a long story short, a further and extensive operation upon the loin revealed a collection of pus very deeply seated, and some caries of the lower dorsal vertebræ. He made a good recovery. I come next to

Group III. Signs and symptoms referable to involvement of the spinal cord.—I must pass briefly over this portion of my subject.

In most cases nervous symptoms occur only in the later stages of spinal caries, and when the nature of the case has already been shown by pain, angular deformity, or other signs. Paraplegia, increase in patellar reflexes, ankle-clonus, decrease in reflexes,—these are the chief indications that the spinal cord is becoming affected.

I suppose I need hardly remind any one here that paraplegia caused by spinal caries is not usually due to the pressure of bone upon the cord. It is due almost always to the accumulation of soft tuberculous matter between the body of the vertebra and the theca vertebralis of the spinal cord. In rare cases it is due to pressure of bone, or to actual involvement of the cord itself by the extension to it of inflammatory processes. When the pressure upon the soft tuberculous matter has been relieved by making the patient lie down, the paraplegia usually passes off. Such has been the history of several of the cases that I show you to-day. Diminution of patellar reflexes is to be expected when the seat of caries is opposite to or below the lumbar enlargement. Increase of reflexes and perhaps ankle-clonus are important signs of pressure upon the cord above the lumbar enlargement.

Occasionally nervous symptoms are among the earliest and most prominent symptoms of a case of spinal caries. I will conclude by relating to you a striking case in illustration of this.

On March 8th, 1900, when I was acting at St. Bartholomew's Hospital for my friend Mr. Bowlby, then absent in South Africa, I was asked by my colleague, Dr. Ormerod, to see a man æt. 37 who had recently been admitted with paralysis of both arms and both legs.

His history was that six weeks previously he had first noticed a gnawing pain at the back of the neck. This pain subsequently passed to the shoulders and arms; and then weakness, first of the arms, then of the legs, set in.

When I first saw the patient there was a good deal of rigidity of the muscles of the neck, but there was no local deformity or tenderness or other definite sign of caries. It was considered doubtful whether the case was one of caries or of new growth.

An exploratory operation was advised on account of the exceedingly critical state of the patient. I removed the laminæ of the fourth, fifth, and sixth cervical vertebræ, and passed a probe round to the front of the spinal cord hoping to find pus. I was much disappointed at finding none, and thought I must have been wrong in believing the case to be one of caries.

Seven weeks later the man died, and it was then found that there was tuberculous caries of the second, third, fourth, and upper half of the fifth cervical vertebræ, with a collection of thick pus pressing upon the spinal cord.

I must now bring this paper to a conclusion. I have not, I need hardly say, attempted to deal exhaustively with this very large subject. I have merely indicated some points which seemed to me to be of importance, and I hope that many other points may be raised and discussed by those here present.

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THREE LECTURES
ON
RESPIRATORY EXERCISES AND PULMONARY PHYSICS.

LECTURE II.

Delivered at the Medical Graduates' College and Polyclinic,

By HARRY CAMPBELL, M.D., F.R.C.P.,
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LADIES AND GENTLEMEN,—In the latter part of the last lecture I dealt with the subject of costal breathing, and I pointed out that there are at least two kinds of costal breathing, that in which the clavicles are raised to the utmost—the clavicular type—and that in which the ribs are raised while the clavicles are kept fixed or nearly so. In both types all the ribs are raised, but in the latter the lower ones chiefly, for which reason we may call it the "lower costal" type. The degree to which the upper ribs move in this second type of costal breathing depends upon the depth of the breathing: if a moderate breath be taken the upper ribs remain comparatively stationary, but in a complete lower costal breath they move decidedly, the inner ends of the clavicles being at the same time carried forward and somewhat upward.

We have now to ask whether any other kind of costal breathing is possible—whether it is possible, *e.g.* for a person to breathe chiefly with the upper ribs while the lower are kept comparatively fixed. Is there, in fact, an "upper costal" type? It is asserted that such a mode of breathing is employed by some singers, but it does not seem to me to be practicable with the normal unsupported chest. Theoretically it is possible, but practically it is impossible, with the *normal* unsupported chest, except, perhaps, as a laboriously acquired feat, to fix the lower ribs and elevate the upper ribs only. When stays are worn, however, this kind of breathing becomes possible; for if, while the lower part of the chest is fixed by tightly fitting stays, a costal breath be taken, expansion is necessarily confined to the upper part; and this leads me to say a few words on the subject of the corset, and its influence on the chest and on the movements of respiration. Unless it is worn so loose as to give practically no support whatever, the corset must of necessity interfere with breathing, and if worn while growth is still incomplete it must interfere with the proper development of the chest also. Suppose the corset is made to fit the chest closely in the position of ordinary mean respiration but without exerting compression, and there are very few corsets which are put on as loosely as this; indeed, I should imagine it practically never occurs, for even those who do not "tighten in" in the ordinary acceptation of the term, yet compress the chest in some degree,—suppose, I say, the corset is worn so loose as just to fit the chest during mean respiration without compressing it, it will nevertheless interfere with adequate expansion of the lower chest, and when the wearer is called upon to take an extraordinary breath she is compelled to expand the upper part of the chest unduly. The only way in which the corset could be worn without interfering with the normal movements and development of the chest would be by fitting it to the chest in the position of extraordinary inspiration, and if that were done it would, of course, be so loose under ordinary conditions as to be an encumbrance. We find in consequence that the thorax of every woman who has worn stays is abnormal, being overdeveloped and unduly mobile in the upper regions—hence the "heaving bosom" of the woman of the novelette. This state of things is pathological; there is normally little or no difference in the breathing of the man and of the woman, such differences as are observed being essentially due to the use of the corset.

I have said that we may regard "upper costal" breathing as impracticable in the *normal* unsupported chest. That there are, however, some corset wearers who are capable, even when the stays are removed, of breathing chiefly with the upper part of the chest, there can be no doubt. I have such a case now under observation in the person of a girl æt. 17, in whom the upper part of the chest moves very freely in breathing, while the lower part is comparatively immobile. This condition must, however, be regarded as pathological.

We come now to the subject of diaphragmatic breathing. The diaphragm springs from the spine, the lower six ribs, and the xiphoid cartilage. From these origins the fibres proceed to be attached to the central tendon; those from the xiphoid cartilage pass almost horizontally back-

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wards, while the remainder travel in a more upward direction, especially those originating laterally and posteriorly. When the diaphragm contracts, three things happen, or tend to happen: (1) the curves of the fibres which, when in a state of rest, are convex (some markedly so) upwards, straighten out; (2) the central tendon is pulled downwards; and (3) the lower ribs are pulled upwards.

In ordinary, quiet, diaphragmatic breathing the first of these three actions is the most pronounced, the central tendon being held in position by the pericardium which is continuous with the deep cervical fascia, and the upward movement of the lower ribs being partially antagonised by the quadratus lumborum and by other muscles which we need not mention. I say partially antagonised, because in an ordinary diaphragmatic breath not only does the abdomen protrude but the ribs are slightly raised. It is, however, possible with a little practice to fix the lower ribs during the contraction of the diaphragm, and this constitutes a purely *abdominal* breath. You see then that a diaphragmatic and a purely abdominal breath are not quite the same. On the other hand, by fixing the central tendon the diaphragm is enabled to bring about by its contraction considerable elevation of the lower ribs. This fixation may be effected by actively retracting the epigastrium by means of the transversales muscles, and thus thrusting up the abdominal viscera against the under surface of the central tendon; and this being a fixed point the whole force of the contraction of the diaphragm is thus spent in elevating the ribs. When the abdomen is retracted and the tendon supported in the way described, it will be found that the lower chest can be expanded considerably, and this is done largely by the diaphragm. If the transversales are then relaxed and the tendon left unsupported from below, the lower chest can be no longer expanded to the same extent as before.

During quiet diaphragmatic breathing the central tendon moves, as I have said, but little, but during a full diaphragmatic breath with flaccid abdominal walls it moves downwards considerably.

The diaphragm is a very powerful muscle. An ordinary individual, who has full control over this muscle, will find little difficulty in lifting a person of his own weight seated upon his epigastrium, as he lies supine, simply by taking an abdominal breath; and I have been struck by the great strength of this muscle even in old and debilitated patients right up to the moment of death. As we shall see, the descent of the diaphragm influences the circulation by pressing the abdominal veins and thus forcing the blood into the right heart, and in the slackening circulation which is wont to herald death one may frequently observe the diaphragm making vigorous efforts to help the failing heart.

The best way to learn diaphragmatic breathing is to lie on the back, to put both hands on the abdomen, and, while concentrating all the attention

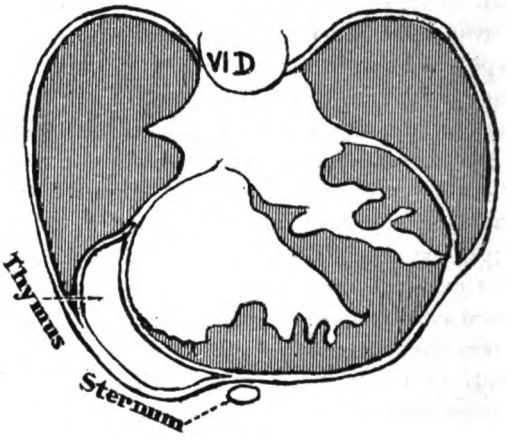

FIG. 1.—Transverse section through thorax of fœtus at the level of the sixth dorsal vertebra. (From frozen specimen.) It will be observed that the solid uninflated lungs lie chiefly at the back of the thorax and only come into limited contact with the heart at its sides.

on this region, to take a breath, at the same time endeavouring to lift the hands by bulging out the abdomen. The same movement should afterwards be practised in the upright position, care being taken to protrude the abdomen during inspiration. When control over the diaphragm has been obtained in this way the diaphragmatic expirations, which ordinarily take place simply by passive recoil, may be aided by active retractions of the abdomen through the instrumentality of the transversales. In this manner the movements of the diaphragm are made to attain their highest amplitude.

I come now to a somewhat difficult subject. Like many others, when once understood it is easy

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enough, but you will, I think, find some difficulty in understanding it from your physiological text-books ; such, at least, has been my case. I refer to the influence of the respiratory movements and the lungs upon the circulation of blood and lymph. This subject is of great importance to the physician, but the lessons which it teaches seem to me to have been largely lost on him. In attempting to elucidate it, and to indicate its applications to medicine, I shall, I feel sure, have your sympathetic indulgence.

The lungs in the fœtus are solid and liver-like. They lie in the posterior aspect of the chest, and only come into limited contact with the heart laterally (Fig. 1). At that most momentous epoch of its career when the child takes its first breath, the chest is expanded in all its diameters—vertical, transverse, and sagittal. The collapsed air-vesicles are pulled asunder, the air being thereby sucked in, and soon all the vesicles are inflated, the lungs having moved the while forwards and downwards so as to occupy the chief part of the thorax and almost entirely to envelop the heart (Fig. 2). I show you on the screen drawings of transverse sections of the chest taken from the frozen cadaver before and after birth, and you will notice the different position of the lung in the two cases. I would like you especially to observe the intimate relation of the right lung to the right auricle after birth ; this relation is admirably brought out in the drawing (Fig. 2), and is full of significance in connection with what I shall have to say later. The left auricle is enveloped by the left lung to a much less extent.

I have said that at birth the air-vesicles are pulled asunder and opened out. To what extent, I would now ask, does this take place ?—a pregnant question, the answer to which is illuminating. It might happen, for aught we could predict to the contrary, that they are opened just sufficiently to admit the air without being put actually on the stretch. But such is not the case : the inspiratory muscles go on contracting, not only until the alveoli are opened up, but *until the whole of the elastic tissue of the lungs is put well on the stretch*, just as you might stretch a piece of elastic. I would like you to bear this fact clearly in mind. And *throughout life these inspiratory muscles make it their business to maintain the lungs in this state of stretch.* The elastic lungs thus stretched are always striving to retract towards their points of attachment, *i. e.* their roots, always striving to escape from contact with the chest wall and other circumjacent structures,—in other words, *the lungs*

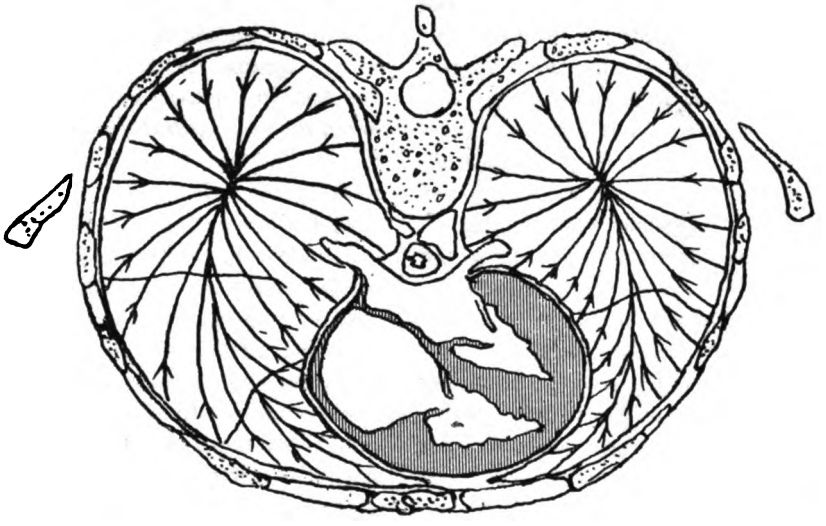

FIG. 2.—Transverse section through the thorax (frozen specimen) after birth, at same level as in Fig. 1. It will be observed that the lungs have moved forwards and now almost entirely envelop the heart. The lungs are intersected with lines marked with arrows in order to indicate how these organs exercise suction on the circumjacent structures. This action can perhaps be the better realised if we imagine each of the lines to consist of a piece of stretched elastic. Note how much more extensively the right lung comes into contact with the right auricle than does the left lung with the left auricle, showing that the former chamber is more under the influence of pulmonary suction than the latter.

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exert suction upon these structures, their pressure being less than that of the atmosphere within them by the contractile elastic force which they exert upon it (see Fig. 2). Note that I use the term *suction*. It is not correct to say that the lungs exercise a *traction* upon the circumjacent structures; inasmuch as they are not physically united to them, their effect is clearly one of suction. We may speak of it as "pulmonary suction."

Let us now inquire into the object of this pulmonary suction. The point I have been endeavouring to bring home is that when the child begins to breathe it so throws its inspiratory muscles into action as not only to open out the alveoli but also to put the lungs on the stretch, and that this results in the lungs exerting suction upon the surrounding structures. Now it is quite certain that the inspiratory muscles would not go to all this trouble of bringing about pulmonary suction without some ulterior object, and we have next to ask what that object is. Pulmonary suction aids expiration, which, as you know, is very largely a passive event. But is this its primary object? Were there time, I believe I could prove to your satisfaction that it cannot be; as it is, I must content myself with saying that I believe the expiratory effect of pulmonary suction to be in large degree accidental. What other structures, then, besides the thoracic walls does pulmonary suction influence? There are the mediastina; above all, the great blood-vessels and the heart to be influenced by it. Has pulmonary suction any special influence upon the flow of blood in the great blood-vessels—the aorta and the large veins within the chest? It has practically no influence upon the aortic flow, nor on that of the great arteries leading out of it; the pressure of blood in them is so great that slight suction leaves them practically unaffected. Does it favour the flow in the great veins of the chest? Certainly it does, its effect being to suck the blood into the venæ cavæ. But I do not think that this can be a primary object, and I will make it clear why I do not. Of the two currents, that in the superior and that in the inferior cava, which is the more in need of help? In the horizontal position of the body the blood flows as readily along the one as the other, but in the upright position the flow in the superior cava is greatly helped by gravity, while that in the inferior cava is as greatly retarded by it. If, then, the object of pulmonary suction be to influence the caval currents, surely it is the inferior and not the superior that ought to be influenced. Now the inferior cava only runs a bare fraction of its course within the chest, while the superior cava and its two great tributaries run the whole of their course there. For this reason we may, I think, safely conclude that the main object of pulmonary suction cannot be to act upon the large intra-thoracic veins. What other important thoracic structure have we left? We have the heart, and I have no doubt that the main object of this pulmonary suction is to facilitate the working of this organ. The lungs almost entirely envelop the heart, and the inspiratory muscles are busy from birth to death in striving to keep up this suction upon it. We may express this fact in another way: *Nature has placed the heart, the great central pump, in a partial vacuum.* What is the object of this? I have here a Higginson's syringe; I squeeze it, and water is driven out; I remove the pressure, and the contracted ball, by virtue of its elastic recoil, gradually expands. Now there is no difficulty about the systole, because it is a muscular act; diastole, however, which is a mere passive recoil, is effected with much greater difficulty, and every now and then you will observe the ball to stick for a moment in diastole, and show a reluctance to dilate promptly. But if this syringe were worked in a partial vacuum diastole would be effected with far greater promptitude than is the case now, the passive recoil of the ball being aided by the suction from without. And so it is with the heart: the heart is placed in a partial vacuum for the express purpose of facilitating diastole, above all auricular diastole. That the ventricles may possess some aspirating power of their own I can well believe, and I would gladly have gone further into this question had there been time; but that the thin-walled auricles must, unless helped from without, often experience considerable difficulty in diastole is self-evident. Especially is this true of the right auricle in the upright position of the body, when the blood in the inferior cava is necessarily flowing at low pressure. You see now why I was so careful to direct your attention to the fact that the right auricle is practically entirely enveloped by the lungs, and is therefore under normal conditions sucked outwards during diastole, thus sucking the blood out of the cavæ—above all, out of the inferior cava, which in the upright position

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of the body is in urgent need of some help to its sluggish flow upward. The left auricle stands in far less need of help from pulmonary suction, and it is of interest to note in this connection that it is much less enveloped by lung than its fellow.

Pulmonary suction is not a constant quantity, but varies according to circumstances. It is largely influenced by the intra-pulmonary air-pressure. Thus during a forcible inspiration with closed glottis there is a considerable fall in this pressure, and the suction action of the lungs is considerably augmented; when, on the other hand, a forcible expiration is made with closed glottis, the intra-pulmonary air-pressure becomes so great as not only to annul all suction but actually to exert a positive pressure on the heart. Concerning the influence of this factor I shall say no more. Another factor determining the degree of pulmonary suction is the extent to which the lungs are stretched: the more they are stretched, the greater the suction; hence, setting aside the influence of the air-pressure, pulmonary suction is at its maximum at the end of a complete inspiration, while at the end of a complete expiration it is entirely annulled, the lungs being then no longer put on the stretch. You therefore see that the position of complete inspiration is the one most favourable to cardiac diastole; then it is that the blood is sucked most forcibly from the cavæ into the right heart, and from the lungs into the left heart. On the other hand, the position of complete expiration is the one least favourable to the diastole of the heart, which in this position experiences no little difficulty in getting an adequate supply of blood from the cavæ and the lungs.

So much for pulmonary suction and its influence on the heart and circulation. Let us now consider the influence on the circulation of the degree of expansion of the lungs. I show you two diagrams bearing upon this matter (Fig. 3). The one represents an infundibulum with its alveoli in a state of extreme inspiration; the other represents the same in extreme expiration. You will observe that in the former condition the lungs contain the maximum amount both of air and blood; in the latter, the minimum amount of each. In other words, when the lungs are expanded to their utmost in inspiration, not only do they contain the maximum quantity of air, but the maximum amount of blood also,—that is to say, in this condition the blood-vessels are opened out to their utmost, the pulmonary circuit offering the minimum resistance to the right heart, and permitting the largest quantity of blood to pass through to the left heart.

But we have just seen that when the chest is expanded to its fullest, pulmonary suction is at its maximum and the heart most favourably circumstanced for sucking blood into its chambers; and therefore we come to the conclusion that *the position of the chest most favourable to the circulation is that of extreme inspiration, and the position least*

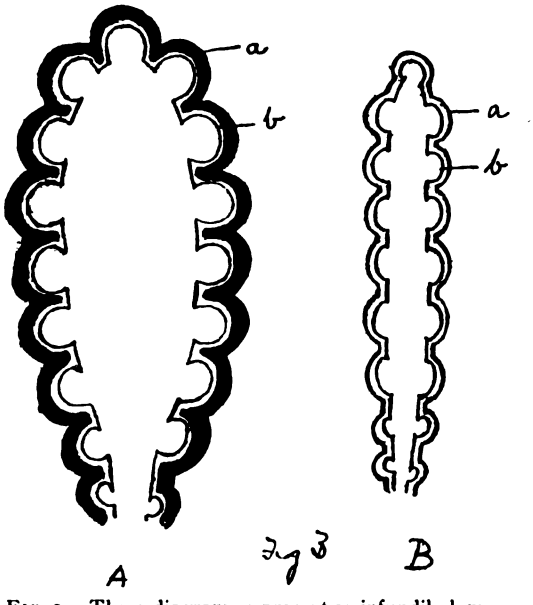

FIG. 3.—These diagrams represent an infundibulum, A, at the end of a complete inspiration, and, B, at the end of a complete expiration. *b* = alveolar wall; *a* = blood-vessels of the same. It will be seen that the amount of blood in the lungs varies directly with the amount of air, and that the pulmonary blood-vessels are most dilated and the resistance to the right heart least at the end of a full inspiration; while, contrariwise, the vessels are most contracted and the resistance to the right heart greatest at the end of a complete expiration.

favourable to it is that of extreme expiration. When I contract my lungs to the utmost I do away with the suction round the heart, because the lungs are no longer on the stretch, and I contract my pulmonary circuit to its narrowest limit and so oppose a maximum of resistance to the right heart, reducing my pulmonary flow to a minimum. When, on the other hand, I hold my chest in complete inspiration, I induce a high degree of pulmonary suction on the heart, thus causing a vigorous flow of blood into it on both sides, while at the same

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time I widen to its utmost the pulmonary bed and thus establish a maximum flow through the lungs. Any of you can test the truth of these statements for yourselves by seeing how long you can hold your breath when the lungs are in a state of complete expiration and complete inspiration respectively. I find I can hold my breath two and a quarter times as long with my chest in extreme inspiration as I can when it is held in the position of complete expiration. Or again, you may make the following experiment:—Take a moderately deep expiration, so as to diminish the mean size of the chest, and then take breaths of ordinary amplitude round that mean. You will find that you soon become dyspnœic.

The position of inspiration being that which is most favourable to the circulation, what should we expect to happen when there is any difficulty in the circulation—when there is dyspnœa, for instance, from heart or lung disease, and urgent need for the largest possible pulmonary flow? We should expect that to occur which we find actually does occur, viz. an expansion of the chest. This I believe to be an important generalisation. It seems so simple and self-evident that it is difficult to believe that it has not been made before, yet, strange to say, I have not been able to obtain any evidence that it has. In all save certain very exceptional cases of dyspnœa, whether physiological or from heart or lung failure, you will find that the individual expands his chest,—that is to say, the mean size of his chest is increased. You will find it expanded in the breathlessness produced normally by exertion, and in that of pathological origin—in emphysema, in bronchitis, in the acute capillary bronchitis of children, in acute pneumonia, whether croupous or catarrhal, in pleurisy with effusion, in pneumothorax, in spasmodic asthma, and in cardiac failure. For some years past I have carefully examined the chest in these various conditions, and I have never failed to find it expanded.

How is the expansion brought about? Obviously by the over-action of the inspiratory muscles. These muscles contract vigorously, just as in a deep inspiration, but they do not completely relax during expiration; hence there is an increase in the mean size of the chest. Were they completely to relax during expiration the chest would return to the position of ordinary expiration. In these cases, with certain exceptions to be mentioned in due course, the expiratory muscles do not come into action at all.

Hitherto we have considered how the *degree of expansion of the lungs* influences the circulation. I have said nothing as to how far the *movements of the chest* influence it. This is another question altogether. The movements of the lungs have, in my opinion, under ordinary circumstances, no influence whatever in accelerating the blood-flow, inasmuch as the increase in pulmonary suction and the width of the pulmonary vascular bed which take place during the inspiratory expansion of the lungs are balanced by a corresponding diminution in each during the expiratory contraction; or, to put it in another way, the mean pulmonary suction and the mean width of the pulmonary bed are exactly the same during inspiration as during expiration. As I read it, then, the mere opening up and contraction of the alveoli have no influence in accelerating the circulation of blood. Were there valves in the pulmonary blood-vessels the case would be otherwise. The lymphatics of the lungs *are* provided with valves, and hence the pulmonary movements quicken the circulation of lymph through the lungs.

While, however, the ordinary movements of the lungs do not quicken the blood circulation, it is possible so to modify them that they shall have an accelerating influence. If, for instance, I take a sudden rapid breath, the sudden inflation of the lung produces an equally sudden negative intra-pulmonary pressure, and this by increasing pulmonary suction will aid the circulation; and if, following upon such an inspiration, I hold my chest for some little time in the inspiratory position, *i. e.* the position most favourable to the circulation, and then allow a moderately rapid expiration to take place, I believe the inspiratory accelerative effect will more than outweigh the expiratory retarding effect. Now this is the form of breathing largely resorted to in physiological breathlessness. If you sprint a hundred yards you will find not only that the mean size of your chest is increased, but that the inspiratory period is more than double as long as the expiratory period.

In the time remaining I will apply these remarks, first of all, to physiological breathlessness; secondly, to emphysema; and thirdly, to heart disease.

First of all, let us consider physiological breath-

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lessness. In all forms of physiological breathlessness the inspiratory muscles are thrown into vigorous contraction, while expiratory muscular action is conspicuous by its absence; and this leads me to say that the best way to develop the lungs is by exercises which induce breathlessness. If you want, *e. g.*, to develop a little girl's lungs, get her to dance and skip; this will make her breathless, the inspiratory muscles will be thrown into vigorous play, and this action will develop the lungs better than any gymnastic exercises with which I am acquainted. No better means can be devised for developing the lungs of children than skipping, dancing, and the running and jumping which find a place in so many outdoor games. Such games are, moreover, far better calculated to develop the physique in a normal manner than the methods by dumb-bells and gymnastics which are now all the rage; the effect of these is to make the individual muscle-bound. As I pointed out in my last lecture, "strong" men have not necessarily good lungs; they increase their chest-girth mainly by developing not the lungs, but the muscles, and, moreover, the development of their thoracic muscles takes place at the expense of thoracic mobility.

Next as to emphysema. This disease is very common: all of us, as we get older, tend to suffer from it more or less; it gives rise to much trouble, and, as might be expected, much has been written about it—and yet, in spite of all this, I do not find that some of the simplest facts concerning it have been properly interpreted. I have not, for instance, met with any satisfactory explanation of the enlargement which the chest undergoes in emphysema, nor of its fixation in the inspiratory position; but if the main conclusions I have put before you are correct, I shall be able to offer you a very simple interpretation of both these features. The chief facts of pathological anatomy to remember in regard to emphysema are—(1) the loss of pulmonary elasticity, and (2) the atrophy of the smaller blood-vessels. As we get older all our tissues tend to lose their elasticity; we see this truth illustrated somewhat painfully, we may think, in the case of the skin: during youth it is closely applied to the underlying tissues, but with advancing years it loses its elasticity and, becoming stretched, is thrown into wrinkles. And just as our skin loses its elasticity, so do our lungs lose their's also. Now if pulmonary suction plays a useful part in the economy—if, as I have endeavoured to show, the inspiratory muscles are, as it were, at great pains throughout life to keep the elastic fibres of the lung on the stretch so as to maintain a certain degree of this suction action,—what should we expect to happen when the lungs begin to lose their elasticity? What but that the inspiratory muscles should come into extra play so as to tighten up the relaxed pulmonary tissue? The pulmonary fibres have, with advancing years, to be strung up continually, even as a stringed instrument has to be from time to time, for otherwise pulmonary suction must steadily grow less and less. Were my lungs at this moment suddenly to grow twenty years older, I am quite sure that my chest would forthwith expand to a notable degree through extra action of the inspiratory muscles, ever on the watch (through their nerve-centres) to maintain pulmonary suction at its normal level. Hence the steady increase in thoracic girth which tends to take place after middle life, or even before it, and the shortening of the neck, as every tailor and dressmaker can testify. Many a man proud of his forty-inch chest would be much better off with a thirty-six circumference. The moderately flat chest with an index of 70, capable of a wide range of movement, is the ideal one. Deepness of chest is a sign of degeneration, and goes along with defective motility.

To return now to emphysema. In this condition the loss of pulmonary elasticity which occurs in all sooner or later, if they live long enough, takes place in an exaggerated degree, and hence the chest tends to be so much the more expanded.

The second anatomical characteristic of emphysema is the atrophy of the small pulmonary blood-vessels. What does that imply? It implies an increased resistance to the flow of blood through the lungs, and a diminished respiratory area. Now we have already agreed that when the lungs expand in inspiration the pulmonary blood-vessels expand at the same time; there is an increase in the amount of blood in the lungs, and a diminution in the resistance of the pulmonary circuit, as well as an increase in the respiratory area. Consequently, as the blood-vessels in the alveoli atrophy, thereby diminishing the respiratory area and imposing increased work on the right heart, the inspiratory muscles come to the rescue and tend to neutralise

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these evils by expanding the chest. There is, therefore, a twofold reason why the chest should be expanded in emphysema—first, because of the loss of pulmonary elasticity; and secondly, because of the atrophy of the pulmonary blood-vessels. This explanation, I trust you will agree with me, is a common-sense one, and I may here say that I have always found common sense to be my best friend in my profession. I have no doubt it is the right explanation. Those given in medical writings—even the most recent works on diseases of the lungs—do not meet the facts of the case.

It is argued that, inasmuch as the elastic recoil of the lungs helps expiration, as the elasticity of the lungs diminishes, the inspiratory muscles, not being properly antagonised, gain the upper hand, and that this leads to an expansion of the thorax—a view first enunciated by Stokes, and accepted by nearly all writers on the subject since his time. This implies that the expansion is a necessary evil and not, as I contend, a cunning device adopted by Nature to counteract certain evils. If it were to the advantage of the emphysematous patient to have his chest diminished instead of increased in size—if such a diminution would facilitate the circulation and the aëration of the blood,—may we not be quite sure that the organism would find means to bring this about? All that would be necessary would be for the inspiratory muscles to contract less vigorously and the expiratory muscles more vigorously than they usually do; *à priori* argument is therefore all against Stokes's view; *à posteriori* argument conclusively proves it to be wrong. I have carefully examined the action of the respiratory muscles in emphysematous patients for some years past, and I have found that while, in the early phases of the disease at least, there is not the slightest tendency on the part of the expiratory muscles to extra action, the inspiratory muscles contract with unwonted vigour. The individual does not expire to the normal position, for the simple reason that he does not want to, his object being to increase the mean size of the chest—with what ends in view I have already indicated. During the more advanced phases of the disease, when the thorax gets fixed and the individual depends mainly upon his diaphragm for breathing, certain expiratory muscles come into extra play, but these are not so much those which depress the thorax as those which push up the diaphragm, for owing to the extreme diminution of pulmonary suction in advanced emphysema the diaphragm is not properly *sucked* up during expiration but has to be *thrust* up by an active retraction of the belly by means of the transversales; and this extra action of the transversales tends to lead to a hypertrophy and shortening of their fibres, and to a narrowing of the costal arch. I have watched this mode of breathing many times.

A few words as to the therapeutic application of these conclusions. First of all, it is manifest that there is no use whatever in applying (as some have recommended) an elastic bandage to the emphysematous chest with a view to make up for defective pulmonary elasticity. The only effect of such a procedure will be to give the unfortunate inspiratory muscles more work to do than they have already, for the emphysematous patient will at any cost keep his chest expanded; but I may here observe that, were Stokes's view correct, it ought to be possible to prevent the emphysematous chest from enlarging by means of such a vest. If you could envelop each lung in an elastic covering, that would be a very different matter, because then you would increase pulmonary suction.

I shall refer to other practical applications in my next lecture, and propose to occupy the time remaining at our disposal by showing you some photographs of patients on the screen.

Cleft Hand.—David and Lipliawsky state that this deformity is a very rare one. Kümmel found but twenty-two cases on record, with forty-two of similar deformity of the foot. The deformity is congenital, and consists in the absence of the middle finger and the corresponding metacarpal bone, the hand being thus divided into two unequal parts. The remaining fingers may be joined together in a firm *syndaktylism*. The deformity is caused by the pressure and traction of amniotic bands *in utero* during the developmental period. One or both hands may be thus affected, and one or both feet as well. It is apt to be repeated in certain families, and males, rather than females, are usually the subjects.—*New York Medical Journal* and *Philadelphia Medical Journal*, July 25th, 1903.

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ON THE ART OF EXTRACTING FOR CATARACT.*

By C. BELL TAYLOR, M.D.,
Surgeon to the Nottingham and Midland Eye Infirmary.

MR. PRESIDENT AND GENTLEMEN,—I have here a patient on whose right eye I operated for cataract in his eighty-sixth year, and on whose left eye I operated for cataract in his ninety-sixth year. As you see, the pupils are central, and he can read for hours and tell the time by the Exchange clock.

I also have here another patient, æt. 60, both of whose eyes I operated upon for cataract by extraction four years ago. On his recovery from the operation he obtained employment as a smith in the furnace department of one of our principal railways, and has since been exposed day by day as a striker to the very trying glare of incandescent metal; nevertheless you will note that both eyes are perfect, that sight is excellent, that he does not require to wear glasses, and that all trace of surgical intervention is conspicuously absent.

The cases of *double* extraction for cataract, to which I called your attention a few evenings ago, although operated on within the last few weeks, were equally remarkable in result and equally free from all trace of operative interference. I propose to-night to lay before you some of the reasons by which I was led to the adoption of a method of treatment which has yielded such satisfactory results.

When Professor Waldau, the late von Graefe's assistant, whose practice I followed in Berlin, proposed to excise a small piece of iris and then to scoop out cataracts with a spoon, he did so because, although at that time suppuration was by no means rare after cataract extraction, it was almost never observed as a result of simple iridectomy. It occurred to me that a slight enlargement of Waldau's incision would enable us to dispense with that very objectionable instrument, Waldau's steep-edged spoon; and later on, seeing the vast importance of maintaining a perfect screen and a central and movable pupil after ablation of the lens, I determined to enlarge Waldau's incision still further, and to dispense not only with the spoon, but also with the iridectomy. Waldau made his incision with a broad, trowel-shaped keratome, entered at the summit of the cornea precisely in the corneo-sclerotic junction. I have attained the same end with greater facility and absolute precision by substituting one or other of the knives depicted in actual size below.

In performing this operation it is necessary to separate the lids with a light speculum, easily displaced; to steady the eyeball with forceps which perforate the sclerotic; and to enter the knife, which should be in first-class condition both as to point and edge, in the cornea, close to the sclerotic junction, at the base of a flap which when completed will comprise nearly, if not quite, one half of the cornea. The knife is then pushed quickly across the anterior chamber until it emerges at a

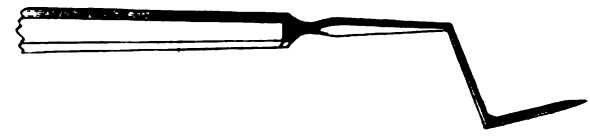

FIG. 1.—For the right eye.

FIG. 2.—For the left eye.

counter-puncture similarly placed; sawed, if I may be allowed the expression, rapidly upwards, so as to get in front of the iris, which would otherwise fold over the edge of the blade; and then turned forwards, so as to complete the section precisely and deliberately in the upper border of the corneo-sclerotic junction. In this way a flap is formed which fits like a watch-glass, so precisely that the wound is invisible the moment the knife is withdrawn, which is at once agglutinated to adjacent tissues, and which ultimately leaves no trace. I prefer to lacerate the capsule widely, but only in its periphery, as suggested by Knapp, and always do this part of the operation with a very light hand, lest the lens be displaced or cortex separated or a portion chipped off. If now slight pressure be made at the lower border of the cornea, or if the patient looks down, the wound will gape, the lens

* Delivered before the Nottingham Medico-Chirurgical Society.

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will present at the pupil, which it slowly dilates, performing a movement of bascule, and then emerges *totus teres;* and if not *atque rotundus*, at all events with an unbroken surface, the flap falls automatically into its place; and if all has gone well there will be no cortex to squeeze out (because there has been none separated from the nucleus), and no prolapse of the iris (because it has not been unnecessarily strained during the passage of the lens), or other trouble then or after to vex the soul of the operator or disturb the serenity of the patient. If the wound does not gape on slight pressure or when the patient looks down—and here is the crux of the whole matter,—it is because

FIG. 3.

the incision is not large enough or the wound has been wasted by cutting between the layers of the cornea instead of entering the chamber at once, in which case it (the wound) may be enlarged by curved, blunt-pointed scissors adroitly manipulated. If it still does not gape on pressure there is some complication or deviation from natural conditions, which will have to be met *secundum artem;* or the eyeball may be closed for a season, and the experience gained be utilised to the benefit of both patient and operator on some future occasion. I will not detain you now with details on these points. What I wish to emphasise is that the operation I have described is differentiated from all others by the extremely narrow knife with a deep shank for the left eye, by which the right hand may be used for the left eye and the necessity for ambidexterity on the part of the operator dispensed with; by the large, easily gaping wound limited to the corneo-sclerotic junction and differing but little in size from Daviel's of one hundred and fifty years ago; by the consequent complete evacuation of the lens and cortex, so obviating prolapse of the iris; and by results which are certainly beyond compare. Indeed, there can be no question that such patients not only look better and see better (the contractile pupil replacing accommodation and compensating, as in aphakial animals, for the loss of the lens), but that they are much better able to bear the brunt of life (witness the smith I have just introduced to your notice), and much less liable to any ulterior ill consequences than if they had been operated on by one or other of the combined methods which have been so fashionable of late years.

When operating I stand behind the patient, who is reclined, and I almost invariably use artificial light. The chimney of the lamp which some of you have seen me use for extraction in this room is an excellent steriliser, and the knives, curettes, and prickers may be soaked in absolute alcohol, while the patient's face, especially the eyelids and roots of the lashes, are sponged with the same fluid.

Cocaine and holocaine in the vast majority of cases suffice for anæsthesia, but if the patient proves very refractory, as now and then happens, ether nebulised by nitrous oxide may be given, or ether alone may be administered by the thermogen inhaler or a modification of the cone, both of which prevent freezing, and are in my opinion far superior to Clover's or any other apparatus of the kind. The patient should be got quickly under by the exclusion of air, and the operation commenced at once during the first period of ether anæsthesia, in which case, according to my experience, you will seldom have sickness or subsequent trouble of any kind.

DR. MAYER, of New York, claims that it is very seldom that the maxillary sinus can be washed out through the natural opening, but that it must be punctured—a very simple matter. He thinks the antrum route to the sphenoid sinus is the best one.—*Med. Rec.*, July 25th.

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A CLINICAL LECTURE

ON

INFLAMMATION OF THE ACCESSORY SINUSES OF THE NOSE.

Delivered at St. Bartholomew's Hospital, June 15th, 1903,

By D'ARCY POWER, F.R.C.S.,

Assistant Surgeon to the Hospital.

GENTLEMEN,—I want to talk to you to-day about the accessory sinuses in connection with the nose,—that is to say, the antrum of Highmore, or maxillary sinus, the sphenoidal sinuses and ethmoidal sinuses, as well as the frontal sinus. Of these sinuses the one in which you will be most interested is the antrum of Highmore. It appears about the fourth month of intra-uterine life, and it may therefore become inflamed soon after birth; whereas with the frontal, ethmoidal, and sphenoidal sinuses any trouble of an inflammatory nature can only be developed at a later period.

I think for your present purposes you may take it that the trouble in connection with the antrum of Highmore is chiefly inflammatory in character. Inflammation occurs under two conditions, for the inflammation is either chronic or it runs a fairly acute course and terminates in suppuration, with or without escape of pus. The pus is not usually pent up, but when it is the symptoms are those which you would expect in an abscess anywhere else, for it is nothing more or less than an abscess inside the superior maxilla. The bone is not very thick, and consequently it may undergo a certain amount of distension, and there is swelling of the face on one side, with considerable pain and œdema. This pain, œdema, and swelling of the face are very often connected with a diseased tooth, because you know that the roots of the teeth open into, or are very closely in connection with, the maxillary sinus, especially the second bicuspid and the first and second molars.

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I need not trouble you very much about the treatment of an empyema of the antrum of this type, because it is so evident. Pus is present, it is pent up, it gives rise to constitutional symptoms, and the patient very soon applies for relief. He usually goes to the dentist, the dentist draws the tooth, and the pus is evacuated. Then the abscess may settle down, and with a little care it all clears up. But there is another form of empyema of the antrum which is much more chronic, and which you are likely to find more troublesome. It opens into the nose, or rather, the natural opening into the nose has never been completely blocked up. Consequently there is a discharge through the nose. The patient does not complain of quite the same symptoms in this case; there is no swelling or redness, and you are very likely to overlook the condition. It is more particularly to these cases that I want to draw your attention just now. You will see on the table different specimens of antral empyemata, and you will have no difficulty in recognising the changes which take place. In connection with these chronic inflammations of the antrum associated with pus, you have to remember that they go on for weeks or months, and that because the opening of the antrum into the nose is not the most dependent part the antrum becomes filled, or partially filled, with pus, and it is only the overflow which is apparent. The patient comes complaining of local tenderness just over the antrum, often in connection with bad teeth, but not always. This is an important point which we shall come to presently. He often suffers from neuralgia, the neuralgia not being localised, as you would expect, to the superior maxilla, but frequently spreading up into the frontal and supra-orbital regions—a distribution which rather leads you to treat the neuralgia and to say it is a referred pain and is connected with the teeth alone, because it does not occur to you to examine particularly the antrum. The neuralgia, too, is often periodical, so that in the case of people living in the tropics the symptoms are apt to be associated with malaria.

The first thing you should do in those cases is to examine the teeth and see whether any are carious. The condition may start from a carious tooth; but on the other hand the diseased tooth may be secondary to the inflammation in the antrum. I mean the inflammation of the antrum has started from some other cause, and those causes, so far as we know them now, are influenza, measles, scarlet fever, typhoid, and tubercle. You must not say, therefore, that every case of antral disease is due to, or starts with, carious teeth. The examination which you should conduct would be first of all to feel over the antrum with the idea of determining whether there is any tender spot. If you found a tender spot you would take the patient into the electrical department and put into his mouth a small electrical lamp, in a dark room, and perform what is called transillumination. When you have him in the dark, in this way you will be able to see whether there is any growth or solid obstruction in the antrum, for on the obstructed side it will appear dark, and the other side will be comparatively light. Thus the transillumination test is an important one in all these cases of doubtful antral disease. You would then examine the patient's nose with a speculum. You may see some drops of pus in the upper part of the middle meatus. If you wipe away the pus and make the patient sit for a time with his head turned so that the affected maxillary sinus is at the highest point, you may see another bead of pus come out just about the same spot. This would be an indication that it was a case of antral or possibly of frontal empyema. The pus in these cases is further characterised by being offensive, and the patient will often come complaining of a bad taste and bad smell. The bad smell is important, because it is the patient who notices it; he will come and tell you that he has some offensive smell in connection with his nose, but his friends do not necessarily complain of it. I have spoken of atrophic rhinitis, and I then told you it was the friends who complained of the smell in that condition and not the patient, because the patient himself has lost his sense of smell in this condition. In both these cases, however, there is offensive pus. You may thus be able to distinguish between patients suffering from ozæna and those who have antral disease. In a given case you know that there is pus present, you have seen a little pus in the nose, and you think it may come from the antrum; the patient is complaining of neuralgia, he has got some tender spot on his cheek, and you find that the two antra are not equally illuminated. You can confirm the diagnosis of empyema of the maxillary sinus by puncturing

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the antrum. An easy way to do this, of course, is to remove the carious tooth, if there is one, and then to push a small trochar and cannula through the socket into the antrum. This simple operation clears up the diagnosis if pus appears. But it does not exclude suppuration in the other sinuses, because empyema of the antrum is often associated with an empyema of the frontal sinus. The two are pretty closely connected anatomically. There may also be a coincident condition of inflammation of the ethmoidal and sphenoidal sinuses, but this is comparatively rare. The treatment resolves itself practically into the treatment of all other abscess cavities,—that is to say, drainage. If you drain the empyema and keep it as clean and sweet as possible, it will recover of itself if you are fortunate enough to see it in an early stage. But when the patient comes for advice the empyema in many cases has been allowed to go on for weeks or months. During this time the patient has not suffered more than discomfort, and there has appeared to be no special reason for interference. The condition is then more difficult to treat. Possibly after the trouble has gone on for a long time the patient has complained of some slight septic troubles: a rise of temperature and a feeling of slackness, loss of appetite or colour, and other indications of septic absorption. Sometimes, when you are at your wit's end to account for these symptoms, you will find an explanation in one or other antrum. In simpler cases the best thing is to puncture the antrum, let out the pus, and then wash out the sinus daily. You can do this with a small Higginson's syringe, using a dilute solution of iodine, or any of the ordinary antiseptics. The little abscess will close up in a short time, and there will be an end to it. This is called the method of alveolar treatment, *i. e.* puncturing the antrum through the socket of the tooth and cleansing it. Supposing that you cannot get sufficient drainage in this way, you may enlarge the opening into the antrum, and keep it patent for a sufficient length of time by putting in a small plug or drainage-tube, the drainage-tube being made of silver wire coiled into a spring, the spring having a flange at one end to prevent it passing altogether into the cavity. But if the disease has gone on for a long time you must do more than this; you may actually have to remove the outer wall by cutting away the bone, and then curette the interior of the antrum with a sharp spoon. When you have scraped away the diseased mucous membrane, you may either plug the antrum or put into it one of the little Ellis's antral drains which I have just described. You may also tap the antrum from inside the nose, but if you adopt this method it is better to remove the anterior part of the middle turbinated bone, and then you can wash out the cavity from the nose. But that is not, I think, quite so satisfactory an operation as that which is done from the mouth. You will see, therefore, that there are three ways of getting at the antrum: from the inside of the nose, from the alveolar border, and from the canine fossa. I would once more remind you that you are extremely likely to overlook the condition of antral empyema with a patent ostium, and therefore I advise you in doubtful cases to go carefully over both maxillary sinuses.

We now pass to the frontal sinuses, which are, in the same way, the seat of two forms of empyema. There is an acute abscess, which usually comes under the notice of the ophthalmic surgeon; and there is a more chronic one, in which the discharge takes place into the nose, and is more likely to come under the notice of the throat department. The causes of an empyema of the frontal sinus are very similar to those which set up inflammation in the maxillary sinus—influenza, injury, and as a sequel of the exanthemata, sometimes also as a result of tuberculous disease. The symptoms of the acute inflammation are œdema, redness, and swelling over one or other of the frontal sinuses, or less commonly over both, situated just above the supra-ciliary ridge, extending downwards into the eyelid. If you cut down upon the abscess and open it in the direction of the eyebrow and drain it for a day or two, the acute abscess may settle down, and there may be no further bad symptoms. But it much more often happens that people come without any external sign of inflammation. They merely say they have a heaviness about the forehead, that it is distinctly tender when they are touched over the eyebrows, and that they have a discharge from one side of the nose. The discharge may pass down the nose and come out of the anterior nares, or it may trickle into the pharynx, and so simulate the cases of naso-pharyngeal catarrh which are of such frequent occurrence in the throat department. The treat-

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ment consists in opening and draining the sinuses. And you need to open them early, because there is a tendency for the pus to track backwards, and instead of passing through the outer table it may pass through the inner table and so set up meningitis. You need not be afraid at the resulting scar which you make, even in a lady, because with a little care the wound heals perfectly, the eyebrow grows over it, and there is scarcely anything noticeable, except to people who look very closely. It is better to shave both eyebrows so that they may grow symmetrically after the operation. You then cut down in the line of the eyebrow itself, beginning at the middle line and making your incision somewhat semicircular. Carry the incision through the periosteum and then open the sinus; cut away the outer table of the skull, and you come down to the sinus almost directly. Put a probe through the lining membrane, and the pus is easily evacuated. When you have got rid of the first gush of pus you had better wash out the sinus with a little boracic lotion, and then take a sharp spoon and scrape gently until you have removed all the granulation tissue. If the operation has been done before, put a probe in and see whether there is any foreign body left from previous operations. I have seen two cases now in which a recurrence of symptoms of inflammation of the frontal sinus was found to be due to the presence of pieces of drainage-tube left from the previous operation. You may drain the frontal sinus in a variety of ways. You may pass a tube down the fronto-nasal canal and drain it through the nostril, or you may make a small aperture in the skin on the inner side of the canthus, take a piece of iodoform gauze through it, and close the wound in the eyebrow, removing the gauze in a couple of days; but the exact method you adopt does not matter very much so long as you keep the sinus clean and dry for a few days. After that you will have little or no further trouble with it, except in those cases where there is tubercle. Tuberculous cases do not lend themselves readily to operation, and they are very likely to become worse as time goes on. We have an old man now in Harley Ward whose frontal sinuses I laid open a fortnight ago for profuse tuberculous suppuration. I do not think we did him the least bit of good, and the suppuration is as bad as ever it was. When suppuration continues in spite of the slighter operation, you must do a more radical operation and remove the whole of the front wall of the cavity, thus allowing the skin and underlying tissues to fall back on the posterior wall and obliterate the cavity. I do not think that is a very good operation, because it sometimes leaves an ugly depressed scar.

Inflammation of the ethmoidal and sphenoidal sinuses is fortunately very much more rare than antral or frontal empyemata. These inflammations may follow the removal of polypi; occasionally they are met with as a result of tuberculous disease. Sometimes you may see them starting as an idiopathic inflammation of the ethmoid bone. They are unsatisfactory, because the sinuses are situated in such close connection with the base of the skull that any inflammation which you find there, especially if it be chronic, is likely to involve the structures at the base of the brain. Thus you may have meningitis, and if so it is usually septic and the patient dies. I have seen two cases of this kind within the last two years; one of them was a case in which a polypus had been removed, and the patient, about forty-eight hours after the operation, suddenly developed symptoms of meningitis, of which he died in a short time. Post-mortem examination revealed the presence of sphenoidal trouble extending backwards, which had evidently been determined by the removal of this polypus. Another case we had here two years ago, in which death occurred after the removal of adenoids. The septic inflammation ran upwards; there was acute inflammation of the ethmoidal and sphenoidal sinuses, and the death of the patient soon followed. In such cases there is very little to be done. The acute cases die, and in the chronic ones you may get an evacuation of the pus by scraping and the very gentle use of a sharp spoon after removing the anterior part of the middle turbinate bone. But in the majority of cases, all that you can do is to keep the patient absolutely at rest, and treat him as you would an ordinary case of meningitis.

Crede's Collargol Treatment.—Sztahovszky ('Pest. Med.-Chir. Presse,' vol. xxxix, No. 17) used collargol in the form of a 15 per cent. ointment in two cases of meningitis, applied three times a day after thorough cleaning of the skin, in order to establish hyperemia. One case (six years old) promptly recovered; the other (four years old) died.—*American Journal of Obstetrics*, July, 1903.

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A LECTURE ON SOME UNUSUAL AUSCULTATORY PHENOMENA MET WITH IN THE EXAMINATION OF THE LUNGS.

Delivered at the Brompton Hospital, June 24th, 1903,

By H. BATTY SHAW, M.D.,

Assistant Physician to University College Hospital, and to the Hospital for Consumption and Diseases of the Chest, Brompton.

GENTLEMEN,—To those of you who give much attention to the physical signs met with in the auscultatory examination of the lungs, it will seem almost impossible to be able to persuade you that anything more can be learned or unlearned by a closer observation of the phenomena discovered to us by the stethoscope. The enormous advantages that have accrued from the systematic examination of the sputa of patients suspected to be suffering from pulmonary tuberculosis are manifold; a few moments spent at such bacteriological examination will save many futile examinations by the older methods. Radiography has also been brought to our aid, and though we are not able, at present, to speak with a certain note of its advantages in the diagnosis of difficult cases of pulmonary tuberculosis, by its means we are able to study the dislocation of the various thoracic viscera in pulmonary disease; and of special importance is the help given in studying the movements of the diaphragm by means of the screen. But just as with the older methods of investigation, these more modern additions have their limitations, *e. g.* the sputum of a tuberculous patient whose lungs are obviously diseased may not show any tubercle bacilli until repeated bacteriological examinations have been made or inoculations carried out; in the same way radiography fails us, and we may not be able to tell how far the heart is dislocated, because the shadow given by it is confused with that of the condensed fibrotic lung in its neighbourhood.

There is therefore every reason to maintain a high standard of accuracy in physical examination by the older methods, controlling them, of course, wherever necessary, by an appeal to the newer methods. It is a common experience in any out-patient room to find cases in which an absence or minimum of physical signs is associated with undoubted proof of invasion of the lung by tubercle bacilli, by the presence of such organisms in the sputum. Further, that *ignis fatuus* of all beginners in the study of pulmonary tuberculosis, viz.: "basal phthisis" may, by bacteriological examination, be shown to be non-tuberculous, or at most, a more recent and active accompaniment of involuting or quiescent tuberculosis of the upper part of the lung of the same side. With such apology for bringing to your notice the desirability of perfecting your auscultatory examination, a consideration may be entered upon of certain phenomena met with amongst the patients now shown for examination.

It is not proposed to weary you with any disquisition upon the particular names applied to various forms of breathing or adventitious sounds; these you have already studied elsewhere, and it is only pertinent to this lecture to bring before your notice some of the recognised rarer auscultatory phenomena, and also some other signs met with amongst the rich material of the out-patient department of this hospital. Before proceeding further, I should like to make full acknowledgement of the great help afforded me by my clinical assistant, Dr. H. M. Page, without whose help and criticism it would have been impossible to collect the cases brought forward for your examination.

1. *Breath-sounds.* — Repeatedly, attention is called by students to the fact that above and below the right clavicle expiration is prolonged or harsh, or that both inspiration and expiration are more marked than usual, and the beginner is loath to pass such a case as sound. Probably this is wise on the part of a student, but if you are going into practice without being aware that, in women especially, the breath-sounds at the right apex are exaggerated quite independently of disease, and possibly dependent upon the more horizontal course and the greater magnitude of the right bronchus combined with the smaller muscular development of women, you will cause much unnecessary anxiety to yourselves and your future patients. "Cog-wheel," "metamorphosed," or "broken breathing," is also a stumbling-block to many, but such breathing is compatible with perfect health, if met with in a nervous subject, on both sides of the chest. When met with at an apex or other part of one lung combined with other signs, as in a case to be shown to you presently, then its significance is very important. This particular patient has pul-

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monary tuberculosis and mitral regurgitation; at the right base behind, inspiration is divided into several parts, and the pitch is altered as the inspiratory phase develops—the sounds are those of very exaggerated cog-wheel breathing, and suggest the possible existence of a cavity or consolidation beneath. Benefiting by the inheritance of Dr. Habershon's out-patients and observations, I am enabled to show you a case—one of bronchiectasis—in which that rare but very remarkable sign known as a "veiled puff" may be heard. It is not possible to enter into the discussion of the definition of this phenomenon as given by Laennec or Skoda, but it may be said that it is considered to be a very suggestive sign of a cavity; you will hear the inspiratory sound suddenly change its character and become both louder and altered in pitch; in this particular patient the sign has been constantly found on all occasions when an examination has been made. It is not necessary to do more than refer to the fact that the breath-sounds in children are often harsher than in the adult, and that with children the so-called "puerile" breath-sounds are the rule. Another difficulty frequently met with is the correct interpretation of bronchial breathing heard at one apex or over one side of the chest when the opposite side is the seat of disease; whether such bronchial breathing is a mere expression of compensatory hypertrophy or of further invasion by disease can only be settled by other signs and a careful repeated examination.

Most students in their second and even in their third year of study, hesitate very much at making a diagnosis of bronchiectasis, and with that humility bred of similar mistakes on my own part, I should like to express my sympathy. You will be able to examine cases this afternoon of bronchiectasis occurring at one base behind, in which there is limitation of movement and dulness to percussion of the apex on the same side, and I hope you will feel thoroughly sceptical of the diagnosis, because it is such a grave mistake to make, to label a case bronchiectasis which is really one of pulmonary tuberculosis. However, the cases shown to you have had the sputum repeatedly examined for tubercle bacilli with a negative result, they have been going on for a considerable time and the patients' general health is much less affected than would be the case were pulmonary tuberculosis responsible for such physical phenomena; lastly, these patients are in the habit of coughing up periodically a large amount of expectoration, which very often is offensive, and still further increased in amount by the assumption of a position likely to empty the cavity into an adjacent bronchus; some of the most marked cases of clubbing of the fingers are to be met with in these cases.

2. *Conduction of vocal and other sounds.*—The majority of patients will reveal the signs known as bronchophony and pectoriloquy on auscultation above the clavicle, especially on the right side, and this fact, combined with the one already mentioned of increased loudness of breath-sounds at the right apex, accounts for the frequent diagnosis of right apical mischief when really there is nothing amiss there; increased vocal fremitus in the same area adds still one more difficulty to the beginner in the diagnosis of apical disease. It is well known that although clubbing of the fingers is met with in several diseases of the lungs, in some forms of heart disease, especially congenital ones, and in certain cases of cirrhosis of the liver, other cases come under observation without any possible explanation being found. There were two such cases amongst the out-patients recently. One remains an enigma to this day; he has but slight cough, very marked clubbing of the fingers, and not a sign anywhere in the chest or abdomen. The other was just as much a puzzle until one day along the course of the right eleventh intercostal space close to the spine behind, pectoriloquy and bronchophony were discovered; the breath-sounds at first were found to be but very little altered, but later on they began to change and now are what may be called bronchial or even tubular in type. This case is mentioned because of the extreme importance of examining the chest repeatedly to its utmost limits in cases of copious or persistent expectoration accompanied by marked clubbing of the fingers, and it also serves to indicate the importance of the use of vocal resonance in such cases. It is well known how changeable are the auscultatory phenomena over a tuberculous or bronchiectatic cavity according as the cavity is full of sputum or empty.

You will find that considerable attention is paid to what are known as "cough phenomena." One of them is the so-called post-tussive "echo" which is simply the sharp conduction of the sound of a

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cough occurring immediately after its production, over lung which is consolidated or excavated. This sign must not be confused with another cough phenomenon, known as a "post-tussive suction-sound," which, though commonly found in cases of excavation, is also to be heard over consolidated lung. You tell the patient to cough and then not to inspire till after a short interval; in this interval immediately after the cough a coarse "sucking" sound is heard.

3. *Adventitious sounds.*—No effort will be made to place before you the ordinary classifications of these sounds, but various phenomena will be shown to you which in their difficulty of interpretation prove such pitfalls to the unwary. It will not be overstating the case to say that those of you who have reached such a degree of skill that you can safely affirm that you hear râles at one apex in a patient who is wasting and has cough, would be satisfied in believing that there was probably pulmonary tuberculosis involving that apex. Unilateral apical catarrhal signs certainly are very suspicious, but you must remember that there are other inflammatory disorders affecting one apex only, and to be met with in patients who are so little affected in their general health as to be able to come up as out-patients for weeks and months together; and the importance of this fact cannot be gainsaid. Such patients are apparently not tuberculous, for their sputa repeatedly examined fail to show tubercle bacilli; only, however, after a vigorous repeated bacteriological examination should such a view be taken of this kind of case, and, as already stated, even then, we cannot be quite reliant on the negative bacteriological results.

Opportunity will be taken this afternoon, however, to show you that there are other phenomena heard at the apices which will always trip the incautious observer, and before discussing them it would be well to define as a râle a short sharp sound forming one of a series; a rhonchus as a longer and continuous sound, either sonorous or sibilant in character.

Two or three patients will be shown to you who have come complaining of cough. Their sputa are free from tubercle bacilli; there are no other signs in the chest than curious coarse rubbing sounds heard over the supra-spinous fossa, most commonly of the left side. This is met with in women mostly, though a patient at University College Hospital, a man, shows it on both sides. This sign was first pointed out by Sir William Gowers in the 'British Medical Journal' of 1876; and he has shown that it can be traced to the shoulder-joint and is due to creaking within it. On several occasions, I have asked gentlemen attending the out-patient practice to express an opinion on this phenomenon, and almost invariably I have been told that, considering that sign alone, the patient concerned must have some catarrhal trouble in the apex observed, or some pleurisy; some called the signs râles, some rhonchi, and others friction. I cannot help suspecting that such sounds are responsible for the diagnosis of that extremely rare condition "apical dry pleurisy," as shown by "apical friction." Duchenne has also described "shoulder-blade friction" due to necrosis, etc., of the scapula, and rubbing of the roughened surface on subjacent structures. Seeing the importance in doubtful cases attached to "one or two" râles at an apex, it is most important to exclude both "shoulder-joint" and "shoulder-blade friction."

Whilst following up a series of cases such as Sir William Gowers has described, certain other phenomena were met with to which I should also like to call your attention. Very frequently cases are sent to the out-patient room here for the mere purpose of an opinion; râles have been heard at one or other apex, and it has been feared that such patients were subject to pulmonary tuberculosis. Some of these cases were apparently quite healthy; they were free from fever or other general symptoms, and the expectoration, such as it was, was quite free from tubercle bacilli. Râles, however, could be heard, and very frequently they were most marked just at the end of deep inspiration, the stethoscope being placed in the angle between the outer border of the sterno-mastoid and the clavicle, or towards the inner end of the first intercostal space near the sternum; in some cases they were heard further out towards the outer part of the clavicle, even in the supra-spinous fossa. On tracing these sounds in various directions they were found at a maximum over the sterno-clavicular or acromio-clavicular joints, and were evidently not due to lung disease at all, *but to certain physical conditions within the joints named*, and were synchronous with movements of these joints. You will

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do well, therefore, in such doubtful cases where apical adventitious sounds are heard, to exclude these "articular" crepitations.

From a prognostic point of view it is of extreme importance to know whether two or more apices or only one is involved. You will notice in one patient shown this afternoon that the very metallic râles heard at the right apex are conducted across the sternum so clearly that the diagnosis was made that the opposite apex was also involved; from evidence derived from the conduction of the sounds and from the absence of other signs at the left apex, it is possible that the left apex is free from disease.

Moist sounds generated in the larynx often obscure the signs produced in the lungs, or even are translated as emanating from the lungs; it is well in such cases to get the patient to clear the throat, but in children afflicted with adenoids and enlarged tonsils there will always be considerable difficulty in getting this done, and a true estimate of the lung condition may not be possible till the pathological condition of the pharynx is remedied by operation.

Another point to which I should like to call your attention is the very frequent diagnosis which is made of "friction" heard in one or other axilla, upon the evidence of sounds heard in the axilla over or near the spot where pain is felt on breathing; sometimes pain is absent, and the signs are still interpreted as friction. It is very important to remember that there may be very extensive dry pleurisy producing very marked friction-fremitus and friction-sound, and yet the patient is quite free from pain, and it is generally conceded that the pleurodynia or pleurisy is more a result of involvement of intercostal nerves than of the pleura. This view is overlooked, and students are so biassed by the idea that two roughened pleural surfaces rubbing on one another must cause pain, that such an assumption outweighs the evidence of their ears, and sounds which are merely varied forms of râles are interpreted as friction instead of being called "adhesion" or "collapse" râles. An illustration will serve to prove this point. A man was admitted to University College Hospital in a somewhat delirious condition; he complained of pain in the neighbourhood of the left axilla on taking a breath; on examining this area, the house physician heard "friction" (?) sounds, and a diagnosis was made of pneumonia involving the left lower lobe. The patient died. Pneumonia certainly was found, not in the area suspected, but at the opposite side of the chest; the two pleural surfaces—visceral and parietal—in the area where the "friction" sounds were heard were found to be adherent, and the adhesions were tough and organised; they could not possibly have rubbed the one upon the other, so that the sounds heard could not have been due to friction. Microscopical sections were made of the adherent pleural surfaces and the subjacent lung. The alveoli immediately under the pleura were considerably collapsed, and it is probable that the sounds heard were due to the opening up of alveoli which by reason of the thickened pleura had been allowed to collapse; hence the use of the term "adhesion" or "collapse" râles. Very similar râles, usually much finer in quality, will be heard in the axillæ in patients who are dying; such signs are associated with œdema of the lung, met with when patients are moribund. As a further help in the interpretation, often a difficult one, of such sounds, it is well to remember that friction can only be heard in recent pleurisy, and is usually unilateral; a chronic inflammatory thickening of the pleura is often associated with synechia of the pleural surfaces, and then "friction-sounds" are impossible. Most observers are sceptical of the value of coughing in such cases, for a cough may just as well modify friction-sounds as crepitations.

4. *Other auscultatory phenomena.*—There are some other signs to which I should like to call your attention. Very frequently a sound, systolic in rhythm, is heard at the margin of the cardiac area or near the ascending aorta. In some cases this sound is due to friction occurring between the pleura covering the pericardial sac and the pleura covering the adjacent lung—the so-called pleuro-pericardial sounds; on other occasions the pulsating heart or aorta appears to drive air in and out of the adjacent lung alveoli, bronchioles, or cavities; such movement of air causes a murmur-like sound, the so-called cardio-pulmonary murmur.

Another murmur which causes trouble is a systolic one heard beneath the clavicles. Sometimes it is met with in cases of apical phthisis; in others it is found in healthy individuals, and is commonly intensified by the assumption of the position of standing at attention, with the shoulders well thrown back; in either case the

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subclavian or axillary artery is so interfered with that the condition of a "vein fluide" is established, and this, as you know, may be accompanied by a systolic murmur.

Recently I saw a young lady who had a large abdominal tumour in the right hypochondrium; the apex-beat of the heart was in the fourth left space in the nipple line, and the upper limit of the liver dulness in the right nipple line, was also at the level of the fourth rib. There were two rounded eminences presenting below the right costal margin, and a loud systolic murmur was heard along a line commencing on the eighth rib internal to and below the right scapula and passing obliquely downwards and forwards to the ninth space in the right anterior axillary line. The diagnosis made was hydatid disease of the liver, but no satisfactory explanation could be given of the murmur at the right base of the chest. At the exploratory operation, nothing but broken-down clot and loose tissues could be discovered after an incision had been made over one of the prominences mentioned above, and the abdominal tumour was found to be solid and not cystic. The final conclusion was that the mass was a soft neoplasm of the kidney and not of the liver, and the murmur heard was the one commonly met with in growths of the kidney and explained by some kink or twist in the renal vessels.

From these remarks you will gather that not only are our recent methods of observation limited and open to error, but also that the older usages have their drawbacks; and I trust that the various difficulties to which attention has been drawn in the auscultation of the lungs, rather than making you hopeless, will show how necessary it is not to rely on one set of physical signs, but to apply all tests and make your deductions from a complete survey.

Adenoma in an Abdominal Scar.—Meyer reports such a case in a woman in whom the right appendages had been removed, and a ventral fixation performed. Two years later a mass connected with the fundus and entering the scar was noted. It was excised and was found to contain epithelial cysts and canals whose origin must have been the serosa of the uterus or the parietal peritoneum.—*N. Y. Med. Journ.* and *Phila. Med. Journ.*, July 11, 1903.

THREE LECTURES
ON
RESPIRATORY EXERCISES AND THORACIC DYNAMICS.

LECTURE III.

Delivered at the Medical Graduates' College and Polyclinic,
By HARRY CAMPBELL, M.D., F.R.C.P.,
Physician to the North-West London Hospital and to the Hospital for Nervous Diseases, Welbeck Street, W.

GENTLEMEN,—In the last lecture I pointed out that the lungs of the fœtus lie at the back of the chest, that at birth they are expanded so as almost entirely to envelop the heart, and that throughout life they are kept stretched by the inspiratory muscles, with the result that they exercise suction on the surrounding structures. I have here a very simple apparatus to illustrate this constant suction action of the lungs upon the heart, and the part played in this result by the inspiratory muscles. [Instrument shown.] Even in the mean position of the chest the lungs are moderately stretched and in consequence exercise suction upon the heart, thus aiding its diastole; during inspiration they are stretched still further, pulmonary suction increasing, and the cardiac diastole being aided to a corresponding extent. I also told you that when the lungs are fully expanded they hold the largest available amount of blood and offer the least resistance to the right heart; and I argued from this that the position of the chest most favourable to the circulation is that of full inspiration, because then the influence of pulmonary suction on diastole is most pronounced, the pulmonary bed widest, and the flow of blood through the lungs most abundant and most rapid. I then went on to point out that when it is desirable to facilitate the circulation, especially that through the lungs, the chest is put in the position of inspiration; that this is the case whether one has to do with simple physiological breathlessness, such as that produced by running and jumping, or with pathological breathlessness, such as that which accompanies heart disease, spasmodic asthma, emphysema, pneumonia, phthisis, pneumothorax, pleurisy, and the like, in all of which cases the chest is found in the position of inspiration. I further pointed out that this position of inspiration is brought about by the predominant action of the

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inspiratory muscles. The only way the chest can be held in an expanded position is by a tonic contraction of these muscles; without this it would, by virtue of its elastic recoil, resume (or nearly so) the position of ordinary expiration directly the inspiratory muscles ceased to act; hence in all these cases the inspiratory muscles come into vigorous action, only partially relaxing during the period of expiration. Lastly, I drew your attention to the fact that the rhythmical expansion and contraction of the lungs, unless modified in a peculiar way, have no influence on the circulation of the blood, although they tend to accelerate the flow of lymph through the lungs. I do not know whether physiologists teach this; indeed, I think they do not. So far as I follow them they teach that the ordinary pulmonary movements do influence the circulation of the blood; according to my reading of the facts these movements have no such influence.

One important fact, however, I neglected to point out in the last lecture—namely, that the rhythmic movements of the diaphragm in respiration materially aid the circulation of the blood. Every time the diaphragm descends it compresses the intra-abdominal veins and tends to squeeze the blood out of those veins into the right auricle at the very time when, owing to the descent of the diaphragm, the suction round that chamber is augmented. Hence the diaphragm plays the part both of a force-pump and a suction-pump. It should be noticed that the blood in the intra-abdominal veins cannot pass into the abdominal walls, or into the lower extremities, because the veins in these parts are provided with valves. You will readily understand that when the movements of the diaphragm are vigorous they must exercise a very considerable influence on the circulation. In extreme failure of the circulation, such as often occurs in the last hours of life, one may frequently observe the diaphragm acting very forcibly, and one can hardly doubt that the main object of this is to hurry on the sluggish blood-stream. I have learned to regard this convulsive action of the diaphragm as a bad sign.

I said just now that "the expanded position of the chest is that which is most favourable to the circulation." This will serve as a text for my remarks this evening. I propose to apply this principle to a number of chest diseases.

Emphysema.—First as regards emphysema. We agreed last time that in this disease there is a loss of pulmonary elasticity and an atrophy of the small blood-vessels of the lung, and that as a result of these changes the inspiratory muscles come into augmented action—first, to secure the necessary degree of tension in the elastic fibres of the lungs, which would otherwise, having lost their elasticity, be relaxed and sinuous; and secondly, in order to open out the pulmonary vessels. Hence we find that the chest in emphysema gets year by year larger and larger, as the lungs get less and less elastic and their blood-vessels more and more atrophied, until in course of time we find it permanently fixed in a position of inspiration. Were I asked to describe the advanced emphysematous chest, I should say that it is fixed in a position of supra-extraordinary inspiration—that it is larger than the normal chest at the end of a complete inspiration. Now we have to ask how it comes about that the emphysematous chest gets *fixed* in this position. This is surely a pertinent question; yet curiously enough it does not appear to have interested physicians much. Of recent books on disease of the lungs which I have consulted, many do not raise it, and but few attempt to explain it. One eminent physician suggests that it is due to some impediment to the egress of air from the alveoli. This view I cannot accept, and for this very simple reason: the emphysematous patient breathes, as we have seen, chiefly with his diaphragm; he takes a deep diaphragmatic breath and then drives up the diaphragm by retracting the belly, in this way forcing the air out of his chest; and seeing that there is no difficulty whatever in getting the air out in this way, it is clear that there can be no great impediment to the egress of air from the alveoli. The reason why he cannot get the normal amount of air out of his lungs is simply because the chondro-osseous walls of the thorax are fixed. But why are they fixed? There are, I believe, two reasons: we have seen that the inspiratory muscles in emphysema are thrown into preponderating contraction, and whenever this happens, whenever a group of powerfully acting muscles is not properly antagonised, they shorten, as is well illustrated in the case of talipes and in many other conditions; consequently, there occurs in emphysema a shortening of the inspiratory muscles, and in this way the thorax gets fixed

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in the expanded position. But a second factor now comes into play; the chest tends to "set," so to speak, in the expanded position—the ribs tend permanently to assume the curvatures of inspiration, the joints to alter in shape, the ligaments to shorten, and so forth.

We may next inquire whether any advantages accrue from this fixation of the chest in emphysema. I have little doubt that it serves a good purpose. We have seen that the maintenance of the inspiratory position is of advantage to the emphysematous patient; now the fixation of the chest in this position necessarily tends to save the inspiratory muscles; without it these muscles would require to maintain a powerful contraction during the period of expiration in order to keep the chest expanded; with it they are, to that extent, relieved of work.

But does the fixation entail also disadvantages? I think that it does. First, it tends to interfere with easy expectoration; patients often experience considerable difficulty in bringing up sputum; this may be because it is very viscid, but it may also result from feebleness of the expiratory blast; in order that the sputum may be brought up easily it is not only required that it shall not be too viscid, but also that there shall be a vigorous expiratory blast; and if the chest is fixed, this latter desideratum is not attained. Another disadvantage attaching to fixation of the chest is the inability to expire deeply; there are occasions when it is of great advantage to be able to take a good deep breath; but this an individual with a fixed chest cannot do, and thus he is handicapped considerably, especially if he develops some acute pulmonary disease, *e.g.* bronchitis or pneumonia, owing to the great limitation of his respiratory range.

For these reasons we should not allow the inspiratory muscles to have it all their own way in emphysema; we should get the patient to practise deep expirations from an early period of the disease; we may order him to take expirations from the mean position of respiration so as to empty his lungs as much as he can; this he should do for ten minutes twice or thrice daily. Secondly, we should advise him to avoid doing anything which is apt to cause breathlessness, which, as we have seen, excites the inspiratory muscles.

Heart Disease.—Let us next consider our principle in relation to heart disease. In most cases of organic cardiac disease the heart is enlarged, sometimes enormously. Especially is this the case with children; I speak haphazard, but I feel sure I cannot be far wide of the mark when I say that it is not rare for a child's heart to occupy as much space as a single lung normally does. Now it is obvious that whenever the heart enlarges there must, unless the chest expands proportionately, be a relaxation of lung tissues. In order, therefore, to keep the lungs at their normal degree of expansion, it is necessary, when the heart enlarges, that the inspiratory muscles shall come into extra play, so as to expand the chest. But there is an additional reason why the chest should then expand: directly there is any failure of the central pump the circulation, especially that through the lungs, wants all the help it can get; clearly, then, it is not enough that the lungs should be expanded to the normal amount; they require to be expanded beyond this, so that the full circulatory advantages attaching to their expanded state may be secured. Hence it is not surprising to find that in all cases of serious heart disease the inspiratory muscles are thrown into violent contraction. In advanced cases what we actually find is this: the patient sits up in bed; he has, as we say, orthopnœa; he fixes his arms and raises his shoulders, so as to get all the purchase he can for the inspiratory muscles attached to the shoulder girdle, in order that he may expand his chest to the utmost. I have no doubt that one of the purposes served by orthopnœa in heart disease—and I should not be surprised if it were the chief purpose—is to give the inspiratory muscles the fullest possible scope, for when the patient is lying down these muscles are placed at a very great disadvantage.

I say the inspiratory muscles in cardiac dyspnœa are thrown into powerful contraction. What about the muscles of expiration? I have carefully examined a large number of cases of heart disease as regards the condition of the muscles of respiration, and I find in most cases the expiratory muscles do not act much, if at all. In children they scarcely act at all, and you can see why; in them the thoracic cage is very pliant, and if the expiratory muscles were to contract vigorously the heart would be subjected to a positive pressure; therefore the child dare not expire vigorously; all his efforts—and it is painful to witness them in bad

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cases—are directed towards maintaining the thoracic cavity at its maximum expansion, so as to keep the lungs well expanded and to protect the heart from the positive pressure to which it must otherwise be subjected. Imagine what would happen if, in an extreme case, the expiratory muscles were to contract powerfully and thus compress the faltering heart and crumple up the overburdened lungs!

When, on the other hand, the chest is rigid, we may sometimes see cases of cardiac dyspnœa in which the abdominal muscles contract with some vigour, but this, I think, is mainly for the purpose of driving up the diaphragm or aiding the circulation in the inferior cava.

Seeing that well-developed, well-expanded lungs facilitate the pulmonary circulation, it is obvious that we should pay due attention to the health and development of these organs in heart disease. I have, indeed, sometimes thought that it is then more important to consider the lungs than the heart itself—in mitral disease at all events. I cannot now enter into the lengthy question of exercise for heart disease, but this much I may say: that exercises which induce a certain amount of breathlessness may benefit by favouring the pulmonary circulation. I do not say that this is the only way in which they may do good.

Pericardial Effusion.—Next let us consider pericardial effusion in relation to our text. As the fluid accumulates in the pericardium the lungs are encroached upon, and in order to get them properly expanded the inspiratory muscles must come into extra action. So long as the effusion is moderate in quantity the lungs may in this way be expanded to the normal, and suction normally maintained, so that no rise in the pericardial pressure occurs; as pericardial distension increases, however, it becomes increasingly difficult to maintain the normal pulmonary suction—although even with a large effusion it may still be possible to maintain some negative pressure in the pericardium,—and sooner or later a time is arrived at when it is no longer possible to get pulmonary suction, the fluid in the pericardium attaining a positive pressure which increases with every increment of the effusion. The heart is now actually compressed—a very serious condition of things. We have seen how great an effort is made throughout life to secure a negative pressure round it; but when the pericardium is distended with fluid under positive pressure, the heart, far from being sucked upon from without, is actually compressed by its enveloping fluid, a serious obstacle being thus opposed to the entrance of blood into the auricles.

The pressure effects of pericardial effusion are usually grouped under four heads:

1. From pressure upon the œsophagus, dysphagia may result. I have never seen an instance of this.
2. Pressure upon the lungs may cause pulmonary embarrassment.
3. Pressure upon the sternum and ribs may, it is said, thrust them outwards.
4. Last, and most important of all, pressure upon the heart impedes its proper action.

With regard to the bulging of the precordium in pericardial effusion, I very much doubt whether this can be due to pressure from within. If the pressure of fluid in the pericardium is sufficiently great to thrust outwards the precordium, it would surely so effectually squeeze the auricles as to prevent the blood from flowing into them, and it seems incredible that the heart could go on beating under such conditions. Therefore, whatever precordial bulging may occur in pericardial effusion I should be inclined to attribute rather to the action of the inspiratory muscles in pulling the precordium outwards away from the heart, so as to minimise as much as possible the pressure upon that organ, than to a thrusting outwards of the chest-wall from within by the distended pericardium.

What are the circulatory symptoms resulting from pericardial effusion? Cohnheim injected oil into the pericardium of animals, and the results he obtained were just what might have been anticipated: the blood-pressure in the veins continued to increase because the blood was prevented from flowing properly into the right heart, while the pressure in the great arteries fell because the blood could not reach them in sufficient quantity. And this is what happens clinically. We find great dyspnœa and distress, distension of the veins of the head and neck, and a feeble pulse, the patient dying from arterial anæmia and passive venous engorgement. You may ask how it is possible for the circulation to be carried on at all when the heart is subjected to a positive pressure from without. The only possible way by which the blood can get into the auricles under such conditions is by being driven along the cavæ under a decidedly

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positive pressure; and the only way to get up a sufficiently high venous pressure for this purpose, as far as I can see, is by means of muscle contractions: the diaphragm and anterior abdominal muscles come into play and exert considerable pressure upon the intra-abdominal venous blood, squeezing it along the inferior vena cava into the right auricle; possibly the cervical muscles have a similar influence on the superior veins.

Phthisis.—Now let us consider the subject of phthisis from the point of view of the principles which we have laid down. By phthisis I mean of course pulmonary tuberculosis; in it the lung structure is corroded and the respiratory area diminished. To make up for this the inspiratory muscles come into extra play so as to expand the unaffected portions of the lungs, and so effectually may they do this that there may be no diminution in thoracic girth, even though there be wide-spread destruction of lung tissue. That is a fact which I do not think is sufficiently recognised. I venture to say that in a very large number of cases of phthisis, especially in the earlier stages, you will find no evidence whatever of contraction of the chest. You may, in fact, get one lung completely destroyed, without any contraction of the affected side; in such a case there will probably be hypertrophy of the opposite lung, and the mediastinum will be drawn over and the diaphragm drawn up. But a patient with advanced phthisis may also show no thoracic contraction, even though no such displacements occur, owing to extreme stretching of the pulmonary tissue and bronchi by the vigorous action of the inspiratory muscles. In this way great dilatation of phthisical cavities as well as of the bronchial tubes and air-vesicles may be brought about, producing, it may be, a condition of bronchiectasis and emphysema.

Contraction of the chest does, however, occur in phthisis, but when this happens it is not, be it noted, by a falling in of the thoracic walls. Such a term suggests that the thorax is normally supported from within, and that in phthisis this support is withdrawn; whereas, as we know, its walls are subjected normally to the suction action of the lungs. The so-called *falling in* of the chest is, of course, a *dragging in* due to the contraction of cicatricial tissue in the pleura and lung. And this leads me to say that the tension of the pulmonary tissue, and therefore pulmonary suction, is (so I believe) augmented in phthisis, partly owing to contraction of the cicatricial tissue in the lungs, and partly to the excessive stretching of the atrophied lungs by the exaggerated action of the inspiratory muscles. Hence the over-action of the inspiratory muscles in phthisis has not (as in the case of most other chest affections in which such excessive action occurs) as one of its objects a maintenance of normal pulmonary suction for the sake of facilitating cardiac diastole: its sole object is to open out the pulmonary blood-vessels and enlarge the respiratory area to the utmost.

The fact that the inspiratory muscles are ever on the alert to maintain an adequate expansion of pulmonary tissue in this disease is one of great practical importance, because it shows us that there is no need to resort to special exercises in it to develop the lungs. The patient himself instinctively secures all the expansion that is desirable, the slightest dyspnœa throwing the inspiratory muscles into exaggerated action.

At a Congress on Tuberculosis, held in London last year, Professor Clifford Allbutt asked whether it might be advisable, in a certain stage of phthisis, to put the patient under respiratory exercises. I ventured to address a letter to the 'Lancet' in response to that question, and with your permission I will read a part of it, because it exactly expresses my views on the subject. "Among the many weighty questions raised by Professor Allbutt in his eloquent address on the above subject, not the least important is that relating to the use of gymnastics in phthisis. After observing that the subject is 'little understood,' and that in the more active stages of pulmonary disease 'gymnastics must be inappropriate,' Professor Allbutt continues: 'In healing stages, when softening has ceased and the lung is drying and laying down protective fibre, may not gymnastics, under supervision as skilled as for cardiac disease, do much to expand and thus to call into healthy function the parts which the tubercle has spared? I seek the answer from those who are dealing daily with these problems.'

"This question is, I think, capable of a decisive answer. In no form of lung disease, not even excepting extensive collapse left by a vanished pleural effusion, are special exercises for the purpose of bringing about pulmonary expansion either needful or desirable. The fact is that in all cases of lung disease sufficiently pronounced to cause

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dyspnœa, the organism of its own accord puts into operation, by virtue of the dyspnœa, a process by which the lungs are expanded just so much as is desirable. Now in phthisis, dyspnœa, even though it should be absent during complete rest, is readily provoked by even moderate exercise, and, therefore, no special exercises are required to promote pulmonary expansion in this disease."

Before leaving the subject I would mention a case bearing upon the same point. Not long ago a medical man wrote in one of the medical journals describing a remarkable result which he had achieved in a case of phthisis by a new method of treatment; he claimed to have increased the girth of the chest in the course of six weeks by three inches. Well, gentlemen, I am quite sure that statement was made in good faith; but I am equally sure it was the result of inaccurate observation; no phthisical chest could be made to expand three inches in six weeks; moreover, even if such expansion could be effected, it could only be by a violent rending of pulmonary tissue at the expense of considerable damage to the lungs. As the result of his dyspnœa the poor phthisical patient's inspiratory muscles are, I repeat, continually thrown into vigorous contraction and expand the lungs just as much as is good for them, and an expansion of three inches beyond this could only take place to the serious detriment of those organs.

Spasmodic Asthma.—Now let us pass on to consider spasmodic asthma and to see if we can apply our principle to it also. During an asthmatic paroxysm the chest is in the position of full inspiration. The shoulders are high; the chest is deep; the diaphragm lies low; further, the breathings are altered in their time relations: instead of inspiration and expiration being as 6 : 7, they may be as 1 : 4. Another feature of the attack is the small amount of air passing into and out of the lungs with each breath.

How are we to account for these phenomena? First, let us briefly look at the pathology of spasmodic asthma. There is no general agreement amongst physicians in respect of it, but this much we may say, that it is either due to a narrowing of the tubes or to an inspiratory spasm. Now in regard to the latter view it is certainly the fact that the chest during the attack is in a position of inspiratory spasm; but such is the case in practically all cases of dyspnœa, as I have again and again insisted, and I think there can be little doubt that this inspiratory spasm in spasmodic asthma is secondary—secondary, in fact, to dyspnœa produced by the narrowing of the tubes. Whether this narrowing is itself due to spasm of the tubes or to swelling of their lining membrane, I shall not stop to discuss—probably each of these factors plays its part,—but this we may, I think, safely predicate: that, given a wide-spread narrowing of the tubes producing dyspnœa, an expansion of the chest necessarily occurs in obedience to the principles I have enunciated, because when only a small quantity of air is permitted to pass into and out of the chest, the distress is far less urgent with an expanded than with a contracted chest, extreme expansion giving the maximum respiratory surface and the maximum pulmonary blood-flow. I made a very simple experiment upon myself yesterday and obtained precisely the result I anticipated. I closed my mouth and partially closed my nostrils so that I was only able to breathe a meagre current of air, and was therefore so far in the position of a patient during an attack of spasmodic asthma. Now what happened? I found my chest gradually expanding and eventually assuming the position of complete inspiration—that position, namely, most favourable to the circulation through the lungs and the aëration of the blood. If a man can only breathe, say, one tenth of the normal quantity of air, he will naturally put his chest in that position which is most favourable to pulmonary circulation and blood aëration, and it is thus I explain the expansion of the chest in the asthmatic paroxysm.

Some other factor must be invoked to explain the prolongation of the expiratory phase, a feature suggesting that there is a greater impediment to the egress than to the ingress of air.

Acute Pneumonia.—Next let us apply our principles to acute pneumonia. In this disease, in croupous pneumonia, for instance, a large number of pulmonary air-vesicles are tightly packed and distended with inflammatory products. As a result a large portion of the lung tissue is placed *hors de combat*, rendered useless as a respiratory medium. But more than this, a considerable part of the pulmonary circuit is blocked by the pressure of inflammatory products upon the small blood-vessels. To make up for this curtailment of the respiratory surface and this narrowing of the

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pulmonary vascular bed, the inspiratory muscles are incited to vigorous action, so as to cause a general expansion of the sound parts of the lung. In this way there occurs an expansion of both sides of the chest. There is, however, a further reason why the chest should expand in pneumonia—namely, for the protection of the inflamed lung; were the chest wall not pulled out by the inspiratory muscles it would press upon the swollen and sensitive lung, and this would hardly be advantageous to the patient. Hence the inspiratory muscles make it their business to pull the thoracic wall away from the inflamed lung, so that it shall exercise no pressure upon it at all. They are not always successful in this, for you may sometimes—though I believe this is rare—find post mortem the surface of the consolidated lung marked with ribs.

Pleural Effusion.—We now come to the subject of pleural effusion. How far does our principle apply here? In this condition fluid is poured out into the pleura and encroaches upon the space which should be occupied by the lung, which thus tends to relax. To counteract this the inspiratory muscles come into extra play, and to such good effect as frequently to maintain pulmonary suction at the normal, even when the effusion is copious, so that you may sometimes get a negative pressure in a pleura containing upwards of 100 ounces. Sometimes, however, quite a small pleural effusion proves, when tested, to be under a positive pressure; and such is always the case with empyema, owing, no doubt, to the agglutination of the lung to the chest wall, so that the fluid being hemmed in, its tension necessarily increases as the effusion goes on increasing.

I think you will agree with me that the fact that a pleural effusion of upwards of 100 ounces may show a negative pressure is a most interesting one; such negative pressure can only be produced by a very powerful contraction of the inspiratory muscles; and in all cases of copious effusions you will find evidence of this: you will find, as might be expected, the chest expanded on both sides—not only on the affected side but on the other also, though rather more, if anything, on the former. It will thus be seen that the expansion on the affected side is not due, as a rule, to the chest walls being pushed out, but to the action of the inspiratory muscles on that side and to their endeavour to secure the normal degree of expansion of the compressed lung.

Catarrhal Pneumonia.—Before passing to pneumothorax let me say a few words on the expansion of the chest in catarrhal pneumonia. If you look at a child in the acute stage of catarrhal pneumonia, or capillary bronchitis—they are practically the same disease,—you will notice that the chest, notably the upper part, is expanded. We have here, again, the same story; the expansion is brought about by the powerful contraction of the inspiratory muscles, and is compensatory. The upper parts of the lungs, being least involved in the disease, undergo the greatest expansion; the lower portions, indeed, may undergo actual collapse (from plugging of the smaller tubes). Hence we frequently find the upper part of the thorax much more prominent than the lower; so greatly, in fact, may the unaffected part of the lung be expanded that the vesicles may be stretched beyond the power of immediate recoil, as shown by the fact that after death the excised lungs maintain this hyperinflated condition. The condition is manifested clinically by the great distension of the chest and by its hyper-resonance on percussion. It is sometimes spoken of as acute emphysema, but it is not a true emphysema, *i. e.* a running together of the alveoli, but merely a hyperdistension of them. Its pathology has been variously explained. To me the explanation of it seems simple, and to be sought in the direction just indicated: it is brought about, I suggest, by the forcible action of the inspiratory muscles; the child instinctively utilises to their full extent all the available alveoli by expanding the lungs to their utmost, in order to get as much air into and as much blood flowing through them as possible; and inasmuch as large tracts of pulmonary tissue are prevented from expanding, the remaining portions are subjected to an extreme degree of stretching by the vigorous action of the inspiratory muscles, so much so that, as I have already said, they temporarily lose their power of recoil.

Pneumothorax.—Finally I ask your attention to pneumothorax as admirably illustrating our principle. By far the most common cause of this condition is the rupture of a phthisical cavity: a small valvular aperture forms in the surface of the lung and overlying pleura, and allows the air to be pumped out of the lung into the pleura during

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expiration, when the intra-pulmonary air-pressure is positive, but does not permit it to be sucked back again during inspiration. Thus, as Samuel West contends, the layers of the pleura are forcibly separated, and the pleural cavity gradually pumped up with air during the expirations. In this way the lung on the affected side gets encroached upon, and may in the end be completely collapsed, for the pressure of air in the pleura, during the expiratory periods at least, will be decidedly positive. The sound lung tends to be encroached upon also, for the mediastinum, being no longer subjected to pulmonary suction on the affected side, is sucked over to the sound side, as well as pushed over by the positive pressure on the side affected. There is thus a great diminution in the pulmonary area, for not only is there in extreme cases complete collapse of one lung, but the other lung is encroached upon. Hence it is imperative that the inspiratory muscles should come into vigorous action in order to get every available air-vesicle well expanded, and every available capillary opened out to its maximum, and what we actually find in these cases is the chest in a position of extreme inspiration or even hyperinspiration. Expansion on the affected side would tend to occur, even without excessive inspiratory effort, from the pumping up of the pleura with air, but as a matter of fact the inspiratory muscles contract powerfully on this side as well as on the other.

As in all the other cases of expanded thorax which we have considered, so in pneumothorax, the breathings are shallow. This may seem strange, but it should be remembered that it is only in this way that the mean size of the chest can be increased: if with the increased inspiratory effort there were a corresponding increase in expiratory effort, the mean size of the chest would remain unaltered, and the advantages which follow upon an increase in the mean would not be secured.

I have now cited several instances of dyspnœa in which the chest is expanded, and did time permit I might add to their number, but I have given enough to illustrate my thesis. I trust I have succeeded in establishing to your satisfaction what seems to me at least to be a not unimportant clinical generalisation, linking together as it does several disjointed clinical phenomena.

I do not wish to imply that in all cases of dyspnœa there is a predominant action of the inspiratory muscles, resulting in thoracic expansion. Cases occur in which the expiratory muscles are thrown into convulsive action, but these cases are peculiar. In whooping-cough, for instance, there occurs a series of expirations, followed, however, by a period in which the chest is greatly expanded by vigorous inspiratory efforts; again, the presence of a foreign body in the larger air-passages, such as the larynx or trachea, may lead to violent expiratory efforts to dislodge it; while, again, the sudden and acute dyspnœa produced in animals by blocking the trachea, or by opening both pleural sacs, leads to expiratory efforts of a convulsive nature. In reference to these latter cases, I may point out that such sudden and urgent dyspnœa would in the ordinary course of nature signify generally the presence of some foreign body in the larger air-passages, and it seems not improbable that the expiratory convulsions which occur in the cases just mentioned may be due to the organism falsely interpreting the cause of the dyspnœa.

In conclusion, ladies and gentlemen, let me thank you for the interest you seem to have taken in these lectures. It has gratified me not a little to have been followed with such close attention.

Post-anæsthetic Brachial Paralysis.—Colton and Allen report four cases of post-anæsthetic paralysis. A cursory search of the literature has yielded only thirty cases of this type. They include in these cases only those in which the paralysis occurred from the position of the arms, and not those where it was due to body-pressure from the lateral position of the patient, or where it was due to an assistant leaning against the patient, or pressure upon the edge of the table. Within a few days there is a beginning return of function, usually in the hand first, then a slower return of power in the extensors. As a rule the sensory disturbances are moderate. Later on soreness of nerve-trunks and of muscles may appear. Most recorded cases have recovered spontaneously, and restoration of function commonly recurred within a few weeks. It is certain that the position of the arms is the cause of the trouble. There is no case recorded in which it occurred where the arms lay on the chest or at the sides of the body, and in all cases where there is a record of the position they were above the head or at least abducted.—*Medicine*, July, 1903.

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THREE LECTURES ON ACUTE APPENDICITIS.

LECTURE I.

ON THE ETIOLOGY, PATHOLOGY, AND SYMPTOMS OF APPENDICITIS IN RELATION TO ITS TREATMENT.

Delivered at the Polyclinic on February 20th, 1903.

By HAROLD L. BARNARD, M.S., F.R.C.S.,
Assistant Surgeon to the London Hospital.

GENTLEMEN,—In this lecture, which is the first of three, we shall consider the causes of appendicitis and such of its symptoms as will guide us in the vexed question between medical and surgical treatment. Next week we shall deal with appendix abscesses.

It has always appeared to me a deplorable thing that when a case of appendicitis has passed through the dangers attending its acute stage in the first week, it should after all succumb to the complications attending the formation of an abscess. In my third lecture I shall deal with general peritonitis, and I hope then to be able to tell you of a course of treatment which we are adopting at the London Hospital which appears to give promise of more hopeful results in this terribly fatal complication.

It is usual to consider an acute attack of appendicitis as the result of quite recent causes. On the other hand, I wish to impress upon you to-day that the conditions which have led to this acute attack have been in process of development for months, or perhaps for years, and that when the circumstances which make the first attack possible have once come into existence the occurrence of subsequent attacks is to be expected. It has been truly said that in the affairs of men all great events are determined by the coincidence of the man and the hour; and in pathology it is equally true that acute inflammations are produced by the advent of

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the micro-organism and its opportunity. The micro-organism in the case of appendicitis is nearly always the *Bacillus coli communis*. The interesting point about this bacillus is that it is a normal inhabitant not only of the human intestine, but of that of nearly all mammals. Yet it is only on very rare occasions that it produces pathological results. This fact is accounted for by the enormous differences which this bacillus exhibits in its virulence. This micro-organism obtained from the normal intestine is found to be of very low virulence when inoculated into animals. When taken from an intestine attacked by catarrhal inflammation it is found to be of much greater virulence; and finally, when derived from cases of abscess and acute peritonitis it is of extreme virulence. This fact is of fundamental importance in the etiology of appendicitis, and we must consider and teach our patients to avoid the conditions which produced these changes in virulence of the colon bacillus. It is held by some pathologists that it is the catarrhal or inflammatory condition of the alimentary canal which increases the virulence of the *Bacillus coli*. On the other hand, some hold that the increased virulence of the micro-organism produces the inflammatory change; but however this may be, the fact remains that when the alimentary canal is attacked by gastro-enteritis the *Bacillus coli* in that alimentary canal is vastly increased in virulence, and is far more potent to attack the point of least resistance—the appendix. Other micro-organisms are occasionally found in connection with appendicitis. First we must notice the *Staphylococcus aureus*, and more rarely the *citreus* and *albus*. The *Streptococcus pyogenes* is sometimes found, and very commonly the *Bacillus aerogenes capsulatus*. This latter micro-organism was first described by Welch, of Johns Hopkins University. As its name indicates, it produces large quantities of gas, and is responsible for the occurrence of emphysema and of the distension of some abscess cavities with gas. This fact has a practical application, for in some cases you will find that although the other signs lead you to suppose that you have to deal with a large abscess, yet on percussing this part of the abdomen it is not only resonant but even tympanitic, and when opened a very considerable quantity of gas will escape with the pus.

The typhoid bacillus has also been found as a cause of appendicitis, and a typhoid ulcer has even been located in the appendix. Still more rarely the *Amœba coli* has produced appendicitis in the course of dysentery, and in a very few cases appendicitis is due to infection with actinomycosis. Some years ago I encountered such a case, where the cæcum and appendix were welded together in a large indurated mass, and although it is impossible to state definitely whether the actinomycosis commenced in the cæcum or appendix, they were certainly both involved at the time of the operation. This case recovered after incision and drainage, and the exhibition of huge doses of potassium iodide.

But if the micro-organism be the real cause of appendicitis it is powerless to produce it without certain conditions being present which we have called its opportunity. Four of these conditions are of common occurrence; they are—(1) the presence of a foreign body in the appendix; (2) the existence of a stricture or kink; (3) an ulcer; or (4) the rich supply of lymphatic tissue in the walls of the appendix, which has led Mr. Bland-Sutton to style it the abdominal tonsil. The rarer conditions which give the micro-organism its chance of attacking the appendix are—(5) trauma, which may be direct or due to the violent action of muscles; (6) the terminal character of its blood-supply; and (7) in women the occurrence of menstruation. We will consider each of these causes in turn.

1. *Foreign Bodies.*—In 60 per cent. of fatal cases of appendicitis a foreign body is found in the appendix. In most cases this foreign body is a small, elongated, friable mass of what at first sight appears to be fæcal matter. When pinched between the finger and thumb it is easily crushed, and it is certain that if its constituents be examined small particles of undigested food will be found; but it is now generally held that a greater part of the material of which it is formed is produced by the inspissation of the secretions of the inflamed appendix.

More rarely we find a definite calculus in the appendix. These calculi are stony hard and covered with little, sharp, nipple-like projections. They are formed of triple phosphates with some organic matter. I have here several such calculi. One you will see is strangely like a date-stone; it was removed from a man who had suffered from

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many attacks of relapsing appendicitis. Others you will see are much larger and more irregular in shape. Their size makes it impossible that they can have entered the appendix from the cæcum. The wall of the appendix is greatly thickened and condensed around them, and the mucous membrane you will see is ulcerated in several places.

True foreign bodies are occasionally found in the appendix. We have here the cæcum, ileum, and appendix of a man who died recently in the London Hospital of general suppurative peritonitis. The end of the appendix is enlarged into an irregular bulbous mass, and projecting from a small gangrenous spot you will see the point of an ordinary pin. I have also seen a grape-stone removed from an appendix, and on another occasion a small shot, which no doubt had been swallowed whilst the patient was eating game.

Recently a good deal of attention has been called to the causation of appendicitis by worms locating themselves in the appendix. These worms are usually the *Oxyuris vermicularis*, whose normal *habitat* is the cæcum and colon. As a cause of appendicitis they must be extremely rare, but a friend of mine recently informed me that he saw an appendix removed for recurrent appendicitis which contained a mass of these little worms twisted together.

These foreign bodies act by harbouring in their substance micro-organisms, and by the mechanical irritation which they produce upon the adjacent mucous membrane. They also obstruct the lumen of the appendix and lead to the retention of secretions in the part beyond.

2. *Strictures and Kinks.*—The lumen of the appendix is often obstructed at or about its base by strictures formed of inflammatory tissue in the submucous layer. In some instances the obstruction is produced by a sharp kink in the appendix, which appears to be due to adhesions in some cases, whilst in others we must rather regard it as a congenital deformity or as a result of the appendix being doubled up in the groove between the cæcum and the abdominal wall. In whatever way the obstruction is produced the result is the same. The secretion of the appendix accumulates in the part of the lumen beyond the obstruction, and dilates it into a considerable cavity. At first the contents of this cavity are clear, gelatinous mucus, and such an appendix is known as a mucocele. In the progress of time this mucus becomes infected with micro-organisms from the adjacent cæcum, and we may then find the dilated appendix filled with pus and its wall ulcerated. This is known as empyema of the appendix.

3. *Ulcers.*—Ulcers are often found in the mucous membrane of an appendix; for the most part they occur in connection with the two previous causes—either a foreign body has irritated the wall of the appendix and produced an ulcer, or the retention of decomposing secretion has led to the same result. But in a few cases ulcers appear to arise independently of these conditions. It is, of course, obvious that such an open sore in the appendix only requires the arrival of the virulent *Bacillus coli* to lead to acute inflammation of the entire structure.

4. *The Lymphatic Structure of the Appendix.*—It is an almost universal custom to speak of the appendix as a vestigial organ, and it is very generally supposed that it is the atrophied end of a cæcum which was very much longer in a previous stage of human evolution. This opinion appears to be incorrect. What its function may be is not known, but it is originally developed in connection with a mass of lymphoid tissue in the wall of the cæcum. This mass of lymphoid tissue is found in all Mammalia, and it is as highly developed in the human being as in any other animal. Mr. Bland-Sutton was one of the first to draw attention to the lymphatic character of the appendix, and from the many close similarities which it presented he styled it the "abdominal tonsil." The submucous tissue of the appendix is always rich in lymphatic tissue, and its end is sometimes converted into a solid mass of lymphoid tissue not at all unlike a lymphatic gland. I have here a diagram copied from the work of Fowler which shows such a specimen in section. You will understand, then, that the appendix will be liable to attacks of acute inflammation in just the same way as the tonsils or the lymphatic glands in the neck, and that just as in children we see acute lymphatic abscesses arise in the neck after a slight catarrhal inflammation of the throat, so an acute suppurative appendicitis may arise from a catarrhal inflammation of the cæcum, or of the mucous membrane lining the appendix itself. I have here a specimen in which the appendix is swollen, congested, and violently inflamed, and yet you will see

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that it contains no calculus, and that there is no sign of a stricture or an ulcer in its lumen.

Turning now to the rarer causes of appendicitis:

5. *Trauma.*—When a disease such as hernia is much more common in men than in women there is a strong supposition that it is due to the more laborious employment of the former. Now appendicitis is four times more frequent in men than in women, and we may therefore suspect that it is often produced by some traumatism. In a few cases the injury to the appendix appears to be direct. There has been some blow upon the abdomen, or the patient has been run over, and the attack of appendicitis supervenes immediately. I have here a specimen which was obtained from such a case. A little boy of ten years old was thrown down in the playground and his abdomen trampled on. He almost immediately complained of acute pain, and the symptoms of general peritonitis rapidly supervened. At the operation no coil of ruptured gut could be discovered, nor were any intestinal contents found free in the peritoneum. The peritoneal cavity was drained as well as his very serious condition would permit, but he died soon afterwards. This specimen, which was obtained at the post-mortem, shows a very large, hard, irregular calculus lying in an ulcerated cavity in the appendix, and you will see at one part a small perforation which produced the peritonitis. We can scarcely believe that this was an example of coincidence.

In another case of mine, a man æt. 35 was kicked in the right iliac fossa by a horse. He vomited fifteen minutes later and developed peritonitis. Some days later a localised appendix abscess was opened, and he recovered. I must, however, confess that the appendix was not actually identified.

Appendicitis very frequently comes on suddenly when the patient is making some unusual or violent effort. A man is lifting some heavy object when he is seized with a violent pain in the right iliac fossa. Several examples have been recorded from the football field. These instances have always been attributed to the action of the psoas muscle. It is supposed that the appendix has become adherent to the sheath of this muscle in some former slight attack, and that when the muscle during the violent movement is contracted to an unusual extent these adhesions are torn across and the lumen of the appendix is opened. No doubt this explanation is correct in many cases, but I wish to offer another possible explanation, and I will tell you how my attention was drawn to the matter. One day, whilst performing an appendicectomy when the appendix had already been removed and its stump was secured in the field of operation by a pair of Spencer Wells' forceps, the anæsthetist allowed the patient to vomit. The root of the appendix was immediately drawn upwards and to the left into the abdomen, a distance of about two or three inches. I immediately pulled it back into the field of operation, and then as the vomiting continued I found that with each spasmodic effort of the patient a very considerable pull was exerted upon the root of the appendix. Now this was not due to any contraction of the psoas muscle, for the thigh was not flexed by the effort. The true explanation is, I believe, as follows:—The root of the mesentery, as you will see from this diagram, passes from the left crus of the diaphragm obliquely downwards and to the right across the spine, and terminates at the ileo-cæcal junction. We are aware that the mesentery has very considerable power of contraction due to the presence in it of the muscle of Lockwood, which is prolonged as the muscle of Treitz into the left crus of the diaphragm; and in violent efforts it is certain that the whole of the mesentery with the attached intestines are drawn up in the abdomen. Mr. Arthur Keith attributes this elevation of abdominal contents rather to the broad band of the transversalis muscle which surrounds the abdomen like a belt. If, then, the appendix becomes attached to the lower extremity of the mesentery and anchors it by adhesions in the right iliac fossa, these adhesions will be liable to be torn during the sudden elevations of the abdominal contents in violent efforts.

6. *Terminal Blood-supply.*—It is a very common experience to find in what appears to be a first attack of appendicitis that the acute symptoms have been produced by sloughing of a greater or less piece of the end of the appendix, and that yet the calculus is situated about the middle of the organ, and that the tissues around this calculus have a good blood-supply and have not lost their vitality. From such cases we must conclude that the chronic inflammation set up around the foreign body has led to the obliteration of the vessels

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running to the part beyond, and that the final thrombosis of these vessels has determined the acute attack. In the work of Fowler, already referred to, there are several beautiful figures of vessels in the mesentery of the appendix in which this obliteration is going on. It is interesting to note, however, that the appendicular artery runs in the free margin of the mesentery of the appendix, and is therefore as far removed as possible from this source of danger. But the mesentery frequently reaches no more than halfway along the appendix, and the artery to the terminal half must then lie in actual contact with the tube exposed to the danger of thrombosis.

7. *Menstruation.*—This is a predisposing cause to which much attention has been directed lately. It is a matter of common clinical experience that attacks of relapsing appendicitis in women are frequently determined by the onset of menstruation. This introduces a difficulty into the diagnosis, for if the attacks be slight and accompanied by insignificant pyrexia the symptoms may easily be due to the congested condition of the right ovary. This connection between menstruation and appendicitis has a most interesting anatomical basis. You will see in this specimen, stretching between the right ovary and the appendix, a well-marked fold of peritoneum which contains a branch of the ovarian artery to the appendix and many lymphatics. It can therefore scarcely fail to happen that if the appendix be in a condition which predisposes it to inflammation more or less acute symptoms should supervene when the ovary becomes greatly congested during menstruation.

You will at once perceive that all these conditions which we have spoken of as forming the opportunity for the micro-organism to attack the appendix were not produced in a day, but required for their development months or even years of preparation. We must, therefore, now consider those insidious and almost symptomless circumstances which lead to the production of foreign bodies, strictures, or ulcers.

First and foremost among these conditions is the narrow lumen of the appendix. In all parts of the body we find that narrow passages, more especially if they be blind, are liable to harbour micro-organisms and to become the seat of chronic inflammation. Thus the follicles of the tonsil become choked with secretion and lead to chronic follicular tonsillitis with enlargement of lymphatic glands in the neck. The lacunæ of the urethra and the glands of the prostate harbour the gonococcus for years, and in a similar way the narrow, long, and tortuous lumen of the appendix is an ideal place where micro-organisms may lurk and secretions may accumulate. This produces a chronic catarrhal inflammation, which, as in the case of the urethra, may easily lead on to stricture when chronic inflammation has once been started in the submucous tissue; or the excessive secretion may become inspissated around some aberrant food particle into a coprolith.

It is almost certain that a chronic catarrhal inflammation of the cæcum precedes that of the appendix. This inflammation in many recurrent attacks invades the narrow lumen of the appendix, and in each of these, as the mucous membrane swells the lumen is obstructed in the same manner as catarrhal inflammation of the duodenum, spreading up the common bile-duct, occludes its lumen and produces catarrhal jaundice.

Now what are the causes which may lead to this chronic inflammation of the cæcum? They are believed to be due for the most part to irregular habits, to meals bolted in haste and without any leisure for digestion afterwards, to an irregular emptying of the bowel; but it is curious that appendicitis does not appear to be due to constipation, for if it were we should expect it to be much more common in women and in older people than in the young. The disease prefers to attack young and vigorous men, who do not as a rule suffer from constipation.

Bad and carious teeth are a very potent predisposing cause of appendicitis. Sir Frederick Treves attributes this to the fact that these people do not masticate their food properly, and that this food in its passage along the alimentary canal tends to ferment and excite catarrhal inflammation. But it appears more probable, in the light of recent work, that carious teeth produce catarrhal inflammation of the whole alimentary canal rather by the condition of oral sepsis to which they give rise. A person whose mouth is in an insanitary condition and many of whose teeth contain large carious cavities is constantly swallowing small quantities of micro-organisms and their products, which, when they have overcome the resistance of the stomach by exciting gastritis, are able to extend their per-

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nicious action further and further down the alimentary canal.

In a very few cases I have seen appendicitis undoubtedly arise in connection with stercoral ulcers in the cæcum due to the presence of large scybalous masses of fæces.

To sum up the etiology and pathology of appendicitis: the conditions which form the opportunity for the virulent bacillus, when once produced, are likely to persist, so that a person who has once had an attack of appendicitis will tend to go on having them until his appendix is destroyed by inflammation or removed by the surgeon. When these conditions are once produced they are a source of ever-impending danger. The mine has been prepared in the insidious stage, and it only requires the spark of the virulent bacillus to ignite it.

The Treatment of Appendicitis.

It must be freely admitted that this is a most difficult and complex subject. In the first place there is probably no disease which is so protean in its form, for in appendicitis we see every degree of severity from a transient pain up to an illness such as fulminating appendicitis, which falls, as it were, from the blue, and destroys a strong man in forty-eight hours. Moreover, whilst the majority of cases appear not to relapse within, anyhow, a limited space of time, many others suffer from periodic recurrences at short intervals, making life intolerable. In collecting the facts upon which we should base an intelligent treatment we also encounter the difficulty that in the specialisation of medical and surgical practice cases of appendicitis are so sorted out that various classes of the profession derive their experience from cases exhibiting very different degrees of severity. The general practitioner sees all the slight and doubtful cases, and unless his practice be a very large one has only a limited experience of the severe forms such as fulminating appendicitis and general peritonitis. Of the more severe cases, the hospital physician receives into his wards only those cases which are considered unsuitable for operation, whilst the hospital surgeon gathers his experience from the most fatal cases of this disease, and in this way no doubt gets an exaggerated idea of its general fatality. If we endeavour to derive our facts from published statistics we cannot but come to the conclusion that these statistics, like other statistics, are quite unreliable. Series of cases published by private individuals would not be published at all unless they were supposed to show a better result than the average, and we must therefore not only be convinced of the honesty of the man who publishes them, but we must know that he has not selected an unusually fortunate series of cases and omitted his less fortunate experience before or after. Fully appreciating and admitting these difficulties, I have endeavoured to glean from the works of all the more important writers on this subject in England and America the fundamental facts which must of necessity guide us in our treatment. These facts are as follows:

1. The attack mortality of all cases of appendicitis, slight or severe, is estimated by Sir Frederick Treves to be about 5 per cent.

2. The attack mortality of severe cases derived from the records of a large number of hospitals, cases which were not operated on, is about 20 per cent.

3. There is no doubt that first attacks are much more fatal, and that they are especially fatal in children. Their high mortality is due to the fact that nearly all abscesses and general peritonitis cases arise in the first attack. Any treatment of appendicitis to be really successful must therefore aim at forestalling and preventing this high mortality at the first appearance of the disease. A line of treatment which allows all severe first attacks to run their course, and is content to stop relapses by an operation during the quiescent stage, can at best only claim to reduce the general mortality 7 per cent., and passively acquiesces in a mortality of 20 per cent. in these severe cases.

4. About a quarter to a third of all cases relapse within a few years. Dr. Hawkins, of St. Thomas's Hospital, found that one quarter of his cases had relapsed, or did so within the few years which had passed since they had been under his care. Fitz found that two out of five of his cases relapsed.

5. It is estimated that these relapses add about 7 per cent. to the general mortality of all severe unoperated cases. This mortality is of importance when considering the question of an interim operation.

6. Fulminating appendicitis, by which we understand cases of appendicitis which die of general peritonitis or septicæmia within three days of the

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onset of the attack, forms about 1 per cent. of mild and 2 per cent. of severe cases. It will be noticed how small this percentage is, and therefore it cannot be used, as it has been, as an argument in favour of promiscuous operation, and certainly not for universal operating in cases after the third day.

7. Probably the most important point in the question of operation is the surgeon, whether he is skilled in abdominal work and has had a large and varied experience of appendicitis, or whether, on the other hand, he is inexperienced, and in the nature of things is not likely to have gained any special knowledge of surgical interference in this disease. I have recently had an opportunity of discussing this question with several American surgeons, and they assure me that in the States there is really a terrible mortality arising from the general belief that anybody, however inexperienced, is competent to operate on all cases of appendicitis at all stages of the disease. The nature of the operation is also an important point when considering the amount of skill required by the surgeon. Localised abscesses when obviously pointing can and should be opened by any qualified practitioner. Acute suppurative peritonitis must be dealt with by the man on the spot, for after all operation in these cases is a forlorn hope, and gives the patient practically his last chance. The interim operation during a period of quiescence does not in most cases require any special experience other than that possessed by any operating surgeon. Operation during the acute stage is, however, a very serious matter, and requires so much experience, judgment, and technical skill that it should only be undertaken by those who have had unusual opportunities of dealing with such cases.

8. The interim operation has a mortality in competent hands of less than 1 per cent. Sir Frederick Treves has published a series of 1000 cases with a mortality of only 0·2 per cent. But this is not a mortality which can be counted on by every ordinary operating surgeon.

9. The mortality of operation in the first twenty-four hours of first attacks is probably 5 to 10 per cent. in the hands of competent surgeons.

10. Operations on the fourth and fifth day (*i. e.* seventy-two to ninety-six hours) have a very severe mortality, 40 per cent. according to Fowler. When the abdomen is opened the suppuration is found not yet to be localised, there is no definite collection of pus to be evacuated, and very few indications as to the extent of the procedures which should be carried out. The gut is often distended, and the manipulations and exposure necessary during the operation greatly increase this distension.

11. Operations on localised abscesses give excellent results.

12. Operations on cases of really general suppurative peritonitis give deplorable results. This latter question is complicated by the great variety of meanings given to the term general peritonitis, but I may say that so great an operator as Sonnenburg, of Berlin, recently admitted that in thirteen cases of general suppurative peritonitis operated on by him the mortality was 100 per cent., and Mr. Rutherford Morison has said that the results are too horrible to be recorded.

These being the facts which must guide us in our treatment, we will now see how they apply to cases at different stages of the disease. The question of how any case of appendicitis should be treated appears to have been confused by the erroneous supposition that we see all cases from the very beginning, whereas in point of fact they come under our care at very different stages. We shall therefore consider the treatment which we should logically follow out at those stages of the disease which appear to be fundamental.

I. *The First Twenty-four Hours of the First Attack.*—The operative mortality in this stage is about 5 to 10 per cent., according to the skill of the operator, and we must balance this mortality against the 20 per cent. which we have seen is that of severe cases in their first attack, *plus* the 7 per cent. which must be added for the relapses to which unoperated cases will be subjected. (*a*) If a case presents mild symptoms, it is better to wait and watch, but the practitioner must fully recognise that every case of appendicitis, however mild, may at any time develop into one of the most severe. A chart should be kept of pulse and temperature, and these should be observed with the abdominal distension at least every two hours. An enema of soap and water, if not too large, will probably do no harm, and relieve the patient by emptying the colon. The question of whether opium should be given at this stage is a very serious one. Considered theoretically it certainly should not, for it most effectually obscures the signs of the development of the disease, and often leads to the serious-

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ness of the patient's condition being overlooked until it is too late to operate with reasonable hopes of success. On the other hand, I fully recognise how almost impossible it is for the general practitioner to refuse to give his patients relief from their severe pain when he and they are aware that many cases so relieved have developed no serious symptoms afterwards. If at any time severe symptoms supervene, or if the pulse, temperature, or distension show that the disease is steadily progressing, then the case should be treated as a severe one and operated on at once. (*b*) Where the onset of the attack is sudden and accompanied by severe symptoms, especially by collapse and great pain, an operation should be performed immediately. It is well known in abdominal surgery that the prognosis of ruptured organs, whether they be the stomach, the intestine, or the bladder, is almost entirely a matter of how soon the operation is performed. This matter may be dogmatically summed up as follows:—If an operation be performed within twelve hours the patient has a good chance of recovery, but if it be performed between twelve and twenty-four hours his chance of recovery or not are about equal; that between twenty-four and thirty-six hours the operation is still just worth doing, but that after thirty-six hours it scarcely comes within the range of reasonable surgery. It must, however, be remembered, as has been pointed out by my friend Mr. Berry, that the appendix is not a capacious organ containing a large quantity of fluid, and therefore its perforation is only in part comparable to rupture of the stomach, gut, or bladder.

II. *Seventy-two to Ninety-six Hours.*—As we have seen, operation during this period is attended with great risk and a severe mortality (40 per cent.). We must, therefore, watch all our patients for at least an hour or so. If the pulse, distension, and collapse, and in a less important degree the temperature, are improving, we may continue to wait and watch. But if the pulse, distension, and collapse are increasing, and the patient's condition is becoming progressively worse, then we should operate, fully recognising, however, that operation in this stage is a most serious matter, and that our manipulations should be as strictly limited as possible, and carried out with all possible speed.

III. *Sixth to the Fourteenth Day.*—Between the sixth and the fourteenth day purulent collections of fluid in the peritoneum become localised into definite abscesses. We have, as a rule, a definite tumour; the pulse remains rapid; in spite of the general improvement in the patient's condition there is often some distension of the intestines, due to obstruction of their lumen where they abut upon the abscess rather than to peritonitis. The temperature also is often somewhat elevated. These abscesses should be opened by suitable incisions as soon as they are diagnosed. But I believe it is a bad practice to search for the appendix in all cases and remove it. This matter will be treated in detail in the next lecture.

IV. *General Suppurative Peritonitis.*—General peritonitis must be operated on as soon as it is diagnosed. We know that general peritonitis is present by the typical facies, the presence of general tenderness and rigidity and the absence of movement over the whole abdomen, by the rapid pulse, which is usually above 130, and by the presence of collapse. A very important sign, to which I think sufficient attention is not paid, is that of the late Mr. Greig Smith. The whole surface of the abdomen is carefully and methodically explored with a stethoscope for a period of at least five minutes, when, if we do not hear the gurgle of any peristaltic movement in the intestines, we may infer that they are paralysed. The operative mortality of general peritonitis is complicated by the very different meanings given to this term by different surgeons. With some all cases of appendicitis seem to be regarded as general peritonitis, but, taking the above signs as our gauge of what is meant by general peritonitis, the mortality appears to vary after operation between 75 and 95 per cent. The conservative mortality may be placed at 99 per cent. The 1 per cent. is reserved for those cases which, after refusing operation, get well in spite of all expectation.

V. *After one Definite Attack.*—There is no doubt that after one definite attack of appendicitis the patient should avoid the mortality of 7 per cent. due to recurrent attacks by having his appendix removed in the quiescent period, with a mortality of less than 1 per cent. in competent hands. He is, moreover, spared the pain, discomfort, inconvenience, broken health, and interference with the business of his life due to these attacks.

VI. *The Treatment of Fistula after Appendicitis.*—These fistulæ are best left alone for at least a

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year. In the vast majority of cases they heal up within that time. They are not a source of danger, but only of inconvenience. Should they not close within this period they may be cautiously enlarged and scraped, and if possible the fæcal concretion which is very probably present should be removed. If they persist for a greater length of time and show no signs of closing, and more especially if recurrent abscesses occur in connection with them, then the patient may be advised to run the risk of a thorough exploration, with removal of the concretion if there be one, or what is left of the appendix.

VII. *The Prophylactic Treatment of Recurrent Appendicitis.*—There are many cases in which the symptoms are so slight that there is considerable doubt as to whether they are suffering from appendicular trouble at all. Some surgeons have styled these cases "appendicular colic." It will often occur that for various reasons these cases are not submitted to operation. How, then, are we to treat them and those patients who refuse operation in spite of the strong advice of their practitioner? In the first place we must see that their teeth are put in thorough order, all stumps removed, and carious teeth extracted or stopped. Deficiencies should be made good by the dentist, and, by means of antiseptic mouth-washes and gargles, the buccal cavity got into as sanitary a condition as possible. Their food should be plain and simple, avoiding such indigestible things as nuts and most vegetables. Their meals should be regular, not eaten in haste, and followed by a suitable period of leisure. If there is any irregularity of the bowels it should be attended to.

VIII. *The Medical Treatment of an Acute Attack.*—It will have been apparent from my previous remarks that I do not regard appendicitis, at least in its severe forms, as a disease to be treated by the physician. But certain cases refuse surgical treatment or cannot obtain it. These cases are best treated by perfect rest. Their diet should be small in quantity and entirely fluid. Purges should not be administered, but, as has already been said, a small soap enema can scarcely do any harm, and will relieve the patient by emptying the colon.

The question of administration of opium has already been touched upon. If for any reason the idea of operation has been finally abandoned, the cases of slight and intermediate severity certainly appear to get through their attack most comfortably with medium doses of opium. But whilst there is the least prospect of an operation none but the very smallest doses should be administered. Finally, I would impress upon you that the most important point in the medical treatment is to watch the course of the symptoms hour by hour in order to form an opinion as to when it may be necessary to call the surgeon in.

SOME PRINCIPLES

UNDERLYING THE

TREATMENT OF EPILEPSY BY PSYCHICAL METHODS.*

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MR. PRESIDENT AND GENTLEMEN,—In a paper published in the 'Lancet' April 18th, 1903, on the treatment of epilepsy by psychical methods, I endeavoured to show that the fact that fits are often modified by various mental conditions may sometimes be turned to practical advantage in treatment. Briefly, the idea brought forward was to encourage and to educate the patient to try to inhibit his fits. The knowledge that fits can sometimes be checked in this way dates from early times, and has often been made use of in cases where an aura ascends the limb, by encircling the limb with a cord and pulling it tight as soon as the aura is felt. Timely stimulation of nerves connected with the special senses will also sometimes succeed. Lastly, the patient himself frequently finds that he has power to inhibit his fits by "fighting against them." This occasional power of inhibition requires further investigation and elaboration, for there is reason to believe that it could often be considerably increased by attention and practice. The difficulty, of course, is to instruct the patient how he is to proceed to help himself, for unless some fairly definite rules can be laid down for his guidance, he will not be much better off than he was before.

So far, I have in my former paper suggested two methods, both of which are very simple.

The first is based on the fact that patients seldom have fits when their attention is fully occupied. They are therefore instructed to perform some particular action with all their might the moment they feel a warning. Naturally, something very simple must be devised, considering the times and places at which fits occur, and the plan I have suggested is that they should grasp one

* Delivered before the East Anglian Branch of the British Medical Association on June 11th, 1903.

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arm as tightly as possible, set their whole mind on the action, and so endeavour to keep off the fit. I have, since trying this, had ample evidence that fits can often be warded off in this way, and have myself had two opportunities of seeing the process successfully carried out.

The second method, which should be carried out as far as possible in conjunction with the first, is to instruct the patient to remember as accurately as possible everything that occurs during the beginning of the fit before consciousness is lost. This idea was based upon the work of Boris Sidis, to which I have more fully referred in my former paper; but, in any case, the effort of memory helps to concentrate the attention at the beginning of the fit.

Boris Sidis found that when patients were under the influence of hypnotism he could obtain from them facts which occurred during the time they were in the unconscious period of the fit; and the beneficial results which he obtained from this form of treatment he explained on the supposition that the fundamental lesion in many cases of epilepsy consists in a functional dissociation of different parts of the nervous system, and that, by regaining the memories belonging to the unconscious period, the dissociated parts are re-associated.

From reading this, it seemed possible that benefit might be obtained by voluntary efforts of memory in the way I have suggested above, although possibly, as I have already said, it may be in a different way to that suggested by Boris Sidis.

Such methods as I have put forward need elaboration, and I hope that by a careful study of different aura further advance may be made.

To-day, in the short time at my disposal, I propose to consider some of the principles underlying epilepsy which make it probable that some cases may occasionally be benefited by these means.

It must be remembered that an epileptic fit is a symptom and not in itself a disease, and therefore, like many other symptoms, it may depend upon a variety of causes. This fact necessitates making a careful analysis of every case, for although it is true that in many instances the ultimate cause will remain unknown, important information bearing on treatment may often be obtained.

If the causes underlying convulsions in general are considered, they may be placed in several groups. For instance, there are (1) convulsions in childhood, (2) convulsions depending upon a specific poison, (3) convulsions associated with gross cerebral disease, and (4) convulsions forming the large group known as "idiopathic" epilepsy.

It is with the problems of idiopathic epilepsy that I now wish to deal, and it is important to consider what, if any, relation this group bears to the other groups; and I shall endeavour to show that a considerable number of the cases in the idiopathic group are probably dependent upon the same causes as those included in the group of infantile convulsions, and, indeed, may be looked upon as an offshoot of the infantile group.

The well-known tendency to convulsions in childhood is considered to be primarily due to an incomplete development of the central nervous system, so that the different parts are not at that time under adequate control. Thus a healthy infant is always more liable to convulsions than a healthy adult, but should, nevertheless, if properly cared for, possess a nervous system sufficiently stable to enable him to pass safely through the trials of that period of his life, unless he is exposed to some unusually severe strain—as, for example, the incidence of an acute specific fever. If, however, the naturally incomplete development is still further retarded by neglect, rickets, or other circumstances, the stability will be lowered in varying degrees, so that any variation in general health, intestinal disturbances, or other forms of irritation will be liable to bring on a fit.

To sum up, then, we may, for our present purpose, recognise three groups of these children:

(1) The healthy infant in whom the relationship between different parts of the nervous system has not yet fully developed, but who still possesses a nervous system sufficiently stable under all ordinary circumstances.

(2) There is the child whose development has been abnormally retarded. Here, with care, convulsions may just be avoided, but the nervous system in this case is very lightly balanced, and a fit will readily occur from local or general disturbances.

(3) There is the child whose nervous system is so unstable that convulsions occur in spite of all care to avoid undue stimulation.

Now, as a rule, the convulsions cease as the

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stability of the nervous system increases with the growth of the child; but in a certain number of cases this desirable stability is never reached, and the child continues to be "subject to fits." It is this class which forms, in many instances, an important link between the convulsions of childhood and the epilepsy of adults; and between the two every gradation may be traced.

It is of course a well-known fact that many adult epileptics suffered from convulsions in childhood, and, indeed, infantile convulsions form a very important predisposing cause of the fits in later life. That there is often an interval of freedom for several years between the fits of childhood and those of later life simply indicates that the relationship between various parts of the nervous system has been strong enough to prevent attacks during the interval of freedom, but has not become firmly enough established to successfully pass through some period during which an extra strain is put upon it—as during the time of puberty, for instance. It is not necessary for the acceptance of this view that fits should have even occurred in childhood. The nervous system may have withstood the early trials, only to break down later, but the underlying conditions may still have been the same. What it really amounts to is the relationship between the nervous system and the impulses it receives. The most stable nervous system may be thrown into convulsions if the stimulation is strong enough—as, for example, in strychnine poisoning. On the other hand, the stability may be so low that the stimulation that would naturally arise during the ordinary course of life cannot be withstood. Whether such stimulation, which is doubtless often the exciting cause of a fit, is the result of auto-intoxication, or of poisons otherwise introduced into the system, cannot now be discussed. The point is that in a large number of cases the nervous system is in all probability primarily at fault, and so unable to resist that which would produce no effect upon a healthy person.

It is especially during the early years of life, when development is still active, that epilepsy first arises. The onset is much less common in later years. Yet it is difficult to imagine that the same "toxins" are not present in later life; indeed, one would expect them to be more plentiful, and most likely they are; but they seldom disturb the equilibrium as they do in youth, although they are harmful enough to cause extensive degenerations. The influence of age is well shown by the following figures taken from 1295 patients admitted into the Ohio State Hospital for Epileptics between 1893 and 1898.*

The ages at which they first suffered were as follows:

In infancy	339
Between 5 and 10 years	194
„ 10 „ 15 „	296
„ 15 „ 20 „	173
„ 20 „ 30 „	159
„ 30 „ 40 „	61
„ 40 „ 50 „	20
Over 50	14
Unknown	39

An interesting paper has recently been published by Dr. Eustace Smith,† in which he draws attention to the possible occurrence of reflex convulsions in growing boys and girls of highly strung and excitable natures. The attacks are often preceded by premonitory symptoms of slight ill-health for several days, and in this way no doubt the balance of their nervous systems, always finely adjusted, is overthrown. Dr. Eustace Smith lays stress on the frequent recovery of such cases, and rightly deprecates classifying them straight away under the head of "epilepsy."

This class of case may, I think, fairly be compared with the second group of infantile convulsions mentioned above, in which the nervous system is just sufficient for ordinary circumstances, but fails when the stimulus is unduly increased, recovering again when the excess of the stimulus is removed, or when a little more stability is gained.

If, then, as it would seem there is evidence to show, the attacks in many cases depend primarily upon an unstable nervous system through feeble or retarded development, it follows that treatment should be especially directed towards remedying such a condition; and it is on these lines that success is being obtained in epileptic colonies. In Letchworth's book, which has already been referred to, the experiences of some of those who have charge of colonies is there given.

Dr. Powell says, "Education in its broadest sense is the chief hope of the epileptic, as it is

* 'The Care and Treatment of Epileptics,' by Letchworth.

† 'Lancet,' January 24th, 1903.

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with the normal child, only modified to suit the strength of the patient." Dr. Spratling says, "No two patients are amenable to precisely the same kind of medical treatment, but in all cases the aim is to build up the physical system by means of proper food, outdoor exercise, and regular habits. Some improve without any other remedy. Others may be suffering from disordered stomachs, chronic diarrhœa, or intestinal trouble, to whom it is necessary to administer drugs."

Trying to inhibit fits in the way I have brought forward is practically a further step in the education of the nervous system. To be successful, constant watchfulness and strict mental discipline between the fits are also required, as well as the greater efforts when the fit comes on. Carefully regulated, continuous occupation is most important, and efforts should be made to repress all "day dreaming" and general slackness, during which the patient falls an easy prey to an attack. I am fully conscious that the value of this form of treatment must necessarily be limited. When the patient is first seen the disease has often become firmly established, and the patient can no more be expected to exercise complete control over the fits than he would be expected to do over any other confirmed habit; but he may nevertheless still often do a good deal, and in spite of difficulties and drawbacks this method will be found useful in conjunction with other forms of treatment, which, needless to say, it should not displace. Its practice increases the confidence of the patient in himself, and tends to produce a more healthy mental condition.

I will quote one case in order to show how patients are sometimes capable of exercising a definite influence on the number of their fits.

A girl æt. 19 recently came under my care at the Hospital for Epilepsy and Paralysis, with a history of fits for eleven years. The fits were very frequent, sometimes as many as nine in a week, and she never went more than a few days at a time without any. She had taken medicine (presumably bromides) for years, and had also been for a time in an epileptic colony.

I gave her some bromide of potassium, and instructed her to do her best to check the fits. She keeps a diary and brings it to me regularly, with an account of the fits and those she has been able to check.

The results for the eight weeks I have had her under observation can be seen by the following table:

	Fits by day.	Fits checked when felt coming on.	Fits by night.
1st week . . .	1	3	2
2nd week . . .	1	3	2
3rd week . . .	2	0	1
4th week . . .	1	1	2
5th week . . .	1	2	2
6th week . . .	1	0	0
7th week . . .	2	2	0
8th week . . .	1	0	0

From this it will be seen that eleven fits were apparently checked, and this is a considerable number, for it makes all the difference whether a patient has one or four fits in a week. The night fits, as of course would be expected, have gone on much as usual. The patient's mental condition has improved, and she now takes part in household work, which she was formerly unable to do.

The line of investigation which seems most likely to be successful is to study the paths which the impulses would most probably take in consequence of any given aura, and then endeavour to check the natural resulting action by some counter-action.

The case I have just quoted will illustrate my meaning to some extent. The aura here is a feeling of fear, and the natural tendency of such a feeling would be to run for help, and in this way the impulses would be flowing down channels of least resistance. The patient in this case had always acted on this impulse, and would run from one room to another to get help the moment she felt the fit coming on, the aura generally lasting long enough for her to get a few yards. To try to counteract this I instructed her not to give way to the feeling and run, but to sit down firmly and endeavour to check the fits as already described.

I do not wish for a moment to give any undue importance to this method, but I bring it before you to-day, because in certain cases it is of use. Anyone who wishes can, with a little trouble, try it without interfering with any other treatment which he may be carrying out at the time.

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TWO LECTURES
ON
SOME PRACTICAL POINTS IN CLIMATOLOGY.

Delivered at the Medical Graduates' College, May 20th, 1903.

By LEONARD WILLIAMS, M.D., M.R.C.P.,
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Editor of the 'Journal of Balneology and Climatology.'

LECTURE II.

ON the last occasion on which I had the pleasure of addressing you, you will remember that we considered the elements which go to the formation of climate, and estimated the degrees of these elements, which permit of the division into "bracing" and "relaxing," with which every one is familiar. A typically bracing climate, you will recall, is provided by a place which is exposed and elevated, which has a low relative humidity, and is therefore liable to rapid and extreme variations of temperature. A typically relaxing or sedative climate is one in which the opposite conditions prevail. It is sheltered and low-lying, with a high degree of relative humidity, and is therefore remarkably equable both seasonably and diurnally. The physiological effects produced by these climates respectively are, in the case of the former, increased metabolism; in the case of the latter, diminished metabolism. In the bracing climate, the human machine is driven at high pressure; in the relaxing climate, the wheels of being are kept going at a pace which is slow. In connection with these differences it is well to remember that a bracing climate is very much more agreeable to live in than one which is relaxing. To use a bicycling simile, it is the difference between coasting down a hill and pedalling along the level. People in good health dislike relaxing climates because such climates demand care in the matter of diet. Metabolism is depressed, so that less food is required, and if people consume their accustomed amount they complain that the place upsets the liver. In a bracing climate, on the other hand, metabolism being increased, both food and alcohol can be consumed with a considerable degree of impunity; and this appeals very decidedly to most people, especially when in good health and in a holiday vein. It is my purpose to-day, in the light of what we have already considered, to touch briefly upon some of the morbid conditions which are amenable to treatment by climate; but before entering into this matter let me remind you that in estimating the effects of what is called climatic treatment, there are many important things to consider besides climate itself. Not the least important among these is change—change of air, change of surroundings, change of diet, change of associations, and even change of language,—and the more complete this change is from the ordinary circumstances surrounding the patient, the more beneficial are your results likely to be. Another element is the opportunity for an open-air life which this kind of treatment affords, with its possibilities for the full utilisation of the remedial effects of sunshine and the obtaining of the exact amount of exercise which is suited to the individual case. Rest from the ordinary routine of life, from business and domestic worries, and from the exigencies of the petty demands of busy lives; all these contribute most powerfully in bringing about those remedial effects which are loosely spoken of as attributable to climate. Having regard to these considerations, it is always necessary to bear in mind that in taking upon yourself the responsibility of recommending a particular climate you ought to be careful to select that climate from among places which are suitable in other respects. Assuming, for instance, that a bracing climate is useful in hypochondriasis, if one were to select a village on the top of a mountain in which to treat the patient, he would in all probability utilise the increased energy which he derived from the stimulating conditions which there obtain for the purpose of cutting his throat. The proper treatment of such a case demands not only a suitable climate but bright society and abundant opportunities for amusement and occupation. I am sorry to have to say it, but the climatic stations and health resorts generally where the greatest efforts are made in the direction of amusement and occupation for invalids are to be found not in this country, but abroad. The municipalities in this country, with very few exceptions, are incurably pig-headed and insular in this matter. While I am on this topic I may as well remind you that one way of occupying both the mind and body of a patient is to subject him to treatment by baths and waters; so that in selecting a station it is often well to combine

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balneological with climatic treatment. Many patients who, under most circumstances, are depressingly recalcitrant in such matters as diet and exercise, become as docile as lambs as soon as they are subjected to the magic ritual of baths, douches, massage, and the other paraphernalia with which bathing establishments and hydropathics are equipped. I repeat, therefore, that in making your selections of climatic stations of whichever sort, do not lose sight of the importance of other factors in what is comprehensively termed climatic treatment, and see to it that the sanitary conditions of the place are above suspicion, that the accommodation is good, that there is enough to amuse and occupy your patient, and, last, but by no means least, that the invalid is placed under the care of a competent and tactful medical man. However excellent the climate itself may be, any shortcomings in any one of these particulars will be recorded against you personally. The discontented invalid does not stop to inquire where the real fault lies, but bestows his blame with impartial brutality upon all concerned.

Let us now return to the question of the proper utilisation of stimulating and sedative climates respectively. The problem, stated broadly, is this: What are the morbid conditions in which we can expect to obtain benefit from climates of either class? And in order to provide an answer I would ask you, What are the morbid conditions in which we habitually prescribe drugs of either class? I have compared a conspicuously bracing climate to ammoniated quinine. Some one else has compared it, not inaptly, to champagne without the subsequent headache; but whichever simile be the more correct, the principle is the same. Now, when you consider the matter, although there are a good many patients for whom we prescribe stimulating and tonic drugs, there are not very many morbid states in which we prescribe them. The vast majority of these patients are suffering from diseases which you could almost count on the fingers of one hand—anæmia, general debility, neurasthenia, dyspepsia, convalescence, certain types of tubercular disease, certain types of goutiness, and the ill-defined states of malnutrition in adoloscents. It would, of course, be possible to extend this list, but in order to do so it would be necessary to introduce subdivisions and saving clauses innumerable. Now the cases which are properly treated by stimulating and tonic drugs are precisely those, and those only, which are properly treated by stimulating and tonic climates. Of those I have mentioned, I would say that slight degrees of general debility, of neurasthenia, convalescence in persons of sound constitution, and malnutrition in children, especially in children of a tubercular diathesis, afford the most typical and conspicuous examples of the conditions in which stimulating climates may be recommended with every confidence of success. The man who is below par and requires a holiday, the man of fairly robust constitution who is convalescing from typhoid or pneumonia or influenza, the weakly anæmic girl with a tendency to enlargement of the glands in the neck; these are people whose vitality wants rousing, whose metabolism wants increasing, in whom you can well afford to drive the machine at high pressure. Remembering that bracing climates are more agreeable to most people than those which are sedative, you will find that patients and their friends will often put in a strong plea for a place where they may be "braced up." This is nevertheless a plea which must be resisted unless you are quite certain that the case is one which demands increased metabolism, one in which it is perfectly safe to drive the machine at high pressure, because by far the greater proportion of patients suffering from disease, as distinguished from minor and symptomatic ailments, require not a bracing, but a sedative climate. This you may take as an absolute rule which admits of few, if any, exceptions. Such exceptions as these are seen in such very protean diseases as tubercle, gout, and asthma. There are doubtless some degrees of phthisis, some types of gout, and several cases of asthma which benefit conspicuously from bracing climates; but the degrees of phthisis are the slighter degrees, and the types of gout the less pronounced types, in which stimulating conditions may be tried without danger; and as to asthma, it is well known to be the most lawless, the most capricious, and the most baffling condition that ever climatologist was called upon to consider. Even in the case of neurasthenia, which I have mentioned as one of the morbid states which may be advantageously treated by stimulating climates, it is of the utmost importance to remember that it is the slighter degrees only

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which are properly so treated. The severer forms have been tersely and quite correctly described as physiological bankruptcy, in which all the vital energies must be nursed and husbanded by every means in our power, in which the very last thing we can afford to do is to drive the machine at high pressure.

Now let us look at the other side of the picture and consider for a moment the morbid conditions which are properly treated by sedative climates; the diseases in which the wheels of being, if they are to be kept going at all, must above all things be kept going slowly; the bodily states in which to drive the machine at high pressure means the rapid exhaustion of the steam and stoppage of the engine. The best and most typical example of this class is afforded by chronic renal disease, whether it be in the form of the large white kidney or the granular kidney. Here the indications are to save the crippled organs as much work as possible by increasing the output of the other emunctories, namely, the bowels and the skin. The bowels may safely be left to the care of the numberless laxatives and cathartics which are open to the diligent practitioner, but here, again, the question of selecting your climate from among the spas which offer special advantages in this respect may profitably engage your attention.

The skin we must educate and encourage to perform the duties which it is now, probably for the first time, called upon to perform, and in this matter, too, the aid of baths, and douches, and massage is by no means to be forgotten or despised. I have already explained that one of the chief characteristics of a sedative climate is its equability, its comparative freedom from seasonal and diurnal extremes, and that this characteristic is invariably associated with a high relative humidity. You will hear some people talk about a climate which is dry and equable. Such a climate, gentlemen, does not exist; and, what is more, it cannot exist, for the reason that dryness and equability are mutually exclusive. It would be as reasonable to talk about a fluid which was acid and yet alkaline. Now this condition of equability, with its attendant high relative humidity, is of the utmost importance in the treatment of chronic renal disease, and both the equability and the high relative humidity have special features of value in this connection. As in chronic heart disease our most solemn warnings to the patient are directed against any sudden or violent muscular exertion, so in chronic renal disease our principal care should be the avoidance of any sudden or extreme change of temperature—the one places an undue strain on the crippled heart; the other imposes an undue burden on the crippled kidneys. That is why equability of temperature is of so much value in these cases. It protects the kidneys more effectually than anything else can do against the strain of rapid and extreme variations of temperature. The reason why its attendant condition of high relative humidity possesses an importance of its own is as follows:—In a dry atmosphere perspiration is increased, but it is insensible; in a damp atmosphere it is slightly diminished in quantity, but it is increased in kind, because it is sensible. Practically the only thing which escapes from the blood during insensible perspiration is watery vapour. Some salts are deposited upon the skin, perhaps, but these are inconsiderable in quantity. During sensible perspiration we rid the system not only of the water itself, but of all the effete matters which it holds in solution; and where we are anxious to spare as much work as possible to the crippled kidneys, these are precisely the secretions which we want above all things to promote. The sufferers from chronic renal disease, therefore, find in a relaxing, equable, humid climate those very conditions which are most beneficial to them, namely, avoidance of extremes of temperature and an increase in the sensible perspiration. I regard chronic renal disease as the condition in which climatic treatment will yield the most gratifying and not infrequently the most astonishing results, and I prescribe relaxing climates for this type of patient with the same confidence and assurance as I prescribe salicylate of soda for the pains of acute rheumatism. As good instances of climates suitable to such cases, let me refer once more to Madeira, the Canary Isles, and Arcachon, abroad; and, at home, the watering places on the south-west coast from Bournemouth to Land's End, remembering about these, that the further west you go the greater the humidity and the greater, consequently, the equability. The place among them with which I am best acquainted, for I used to live there, is Sidmouth, in Devonshire, and, from what I have seen of its influence

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in such cases, I have every confidence in recommending it to your notice.

I have left myself but little time for the consideration of other morbid conditions which are suitably treated by this type of climate, but if you will remember the formula of the test which is to be applied in every case which presents itself to you, you will have no difficulty in arriving at a just conclusion in the majority of instances. This formula is, "Can we afford to drive the machine at high pressure, or must we nurse the vital resources?"

I have already incidentally mentioned heart-disease. Now, in view of our formula, it is hardly necessary for me to say that this is a condition which is not suited to bracing climates of any sort, and that it is eminently unsuited to climates of high altitude. These cases are now very frequently sent to spas where the Nauheim treatment is to be obtained, and although this form of treatment may have had too many claims put forward on its behalf, there can be no doubt whatever of its value in suitable cases. But if these patients are to enjoy the maximum of advantage from the treatment, the spa should be selected from among those situated in sedative climates. In this country all these advantages, both balneary and climatic, are to be obtained at Bath, in Somersetshire, where not only is the climate suitable, but the baths are among the most luxurious and best equipped in Europe.

Of functional nervous diseases it is only necessary for me to mention epilepsy, hysteria, and insomnia (if, indeed, the latter can properly be called a disease) for any one to say at once to which type of climate such cases should be sent. These are emphatically conditions which no sane man would treat by stimulation; indeed, every one, or nearly every one, treats them all with bromide mixtures, and it is to the climatic representatives of bromides, namely, humid relaxing stations, that the climatologist directs those suffering from such complaints.

In organic nervous disease, such as tabes, G.P.I., disseminate sclerosis, and the like, we have for the most part diseases which though unhappily incurable, are nevertheless seldom rapidly fatal, and if we are careful to "keep the wheels of being slow" and refrain from any attempt to "brace up" the patients, we can, by placing them under favourable climatic conditions, removed from business worries and the wear and tear of life in large towns, do much to render their lives not only longer, but more enjoyable and more peaceful. In relaxing climates you will find such patients undergo very considerable subjective improvement; in bracing climates they invariably do badly.

Among other diseases which are properly treated by sedative climates, it is well to lay stress upon bronchitis and emphysema. In acute bronchitis we moisten the air of the sick-room artificially by means of the bronchitis-kettle, and very grateful to the irritable air-passages such a proceeding invariably proves. In chronic bronchitis the moisture is equally grateful and beneficial, and can very easily be obtained in the humid climates of our south-west coast stations, where it is to be found combined with that purity of atmosphere which is so conspicuous by its absence in the ordinary sick-room. In emphysema we have in the lung a condition which is exactly comparable to the state of the kidney in chronic interstitial nephritis, and you will find a sedative climate as beneficial in the one as you will in the other. As long as you do not ask too much of the crippled organs by any attempts to drive the machine at high pressure, you can ensure to these sufferers both extended days and comparative comfort. Under functional nervous conditions I ought perhaps to have included the many curious and unpleasant subjective sensations from which women suffer at the menopause. Such patients should never be stimulated, and if you can get them away from their ordinary surroundings to a sedative climate, you will do more to relieve their sufferings than by any drug in the pharmacopœia, with the single exception of icthyol, which seems, indeed, to be something in the nature of a specific in this condition.

Finally, I need hardly say that old people are not among those in whom you should make any attempt to drive the machine at high pressure. In a sheltered relaxing valley near Sidmouth there is a small village called Sidbury, of which it is said by the natives that he who takes up his residence there may live as long as he likes; I am not quite sure about the living, but I can testify to the fact that he may exist for a number of years, which to most of us seems very astonishing. Old people are certainly better away from large centres of busy life, and the best places for them to go to are the warm, equable, sedative stations, where there is plenty of sunshine. I fear that the little which I have been able to compress into these two lectures on this very important and too little appreciated branch of therapeutics has been barely sufficient to provide even a sketch of the general principles of the subject; but whether or not I have contributed anything to the understanding of these general principles, I would like, before we part, to refer once more to a matter of detail, which is this: Whatever other mistakes you may be guilty of, do not, I beg of you, increase the counts of the indictment which the Recording Angel may have against you by sending your chronic renal cases to a bracing climate.

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A DEMONSTRATION OF CASES OF SKIN DISEASE.

Delivered at St. Bartholomew's Hospital, July 6th, 1903,

By J. A. ORMEROD, M.D., F.R.C.P.,

Assistant Physician to the Hospital.

GENTLEMEN,—To-day I propose to show you cases instead of giving you a set lecture. I am afraid that they do not form a properly connected series, but you will excuse this, because it is difficult to get such a series of patients to attend at any one time. Last time, you will remember, I lectured on the subject of bullous diseases, and I described to you the disease known as "pemphigus vulgaris." I could not at the time illustrate it by a case, but since then one has come into the wards, and I have had him brought down for your inspection. The patient is a man somewhat over fifty. He has been in the hospital since June 16th, and when he came in his forearms and legs were covered with bullæ. Unfortunately for the purposes of demonstration, these have now largely cleared up, but you can see here one fresh one about twice the size of a pea. The original bullæ were as big or rather bigger than a sixpence. Some of them contained clear serum, others blood-stained serum. They either broke, or dried up without breaking, but left no open sore and no scar, only these reddened and slightly raised areas which you see. One of these areas is, you may notice, slightly pigmented. The bullæ appeared to arise on normal skin; the reddened marks which I have pointed out to you are the remains of them. The serous fluid from one of them was examined by Dr. Jeudwine, my house physician, and he found it contained a large number of eosinophile cells, as is usual in this disease. Before admission this patient was treated with a mixture of iron and nux vomica, but apparently without much result. After admission we added to his draught ℥iv of liquor arsenicalis,

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and he was given this medicine three times a day. Whether as the effect of the arsenic, or of the better conditions of his life in hospital, I cannot tell you, but the bullæ soon ceased to appear. Then, in consequence of diarrhœa, he had to leave the medicine off for a few days, and a relapse occurred. Perhaps too much stress should not be laid on this occurrence, for the bullæ of pemphigus are apt to appear in successive crops. There is no definite and constant cause common to all cases of pemphigus. This particular patient suffers from malignant disease. I do not, of course, assert that this has any special connection with his skin disease, though conceivably it may have. Here you can see a prominent mass of new growth at his umbilicus. This has been present since August last, and he has been advised that a surgical operation on it would be useless. Nevertheless, while under treatment for his skin disease Dr. Walsham has promised to see what can be done for it by the X rays.

The next cases are rather peculiar. I mentioned to you last time that there is a form of bullous disease, probably not related to common pemphigus, called cheiropompholyx. Its distinctive features are that it affects the hands and feet, on which vesicles and bullæ appear, which soon clear up, but which have a tendency to recur; and that it usually occurs in the summer time, and in people who sweat profusely. I showed you also another variety of bullous disease, which is congenital, or even hereditary, in which, from the patient's infancy, bullæ filled with serum or blood are easily produced by any slight friction or blow. This disease is called "epidermolysis bullosa," or sometimes "congenital pemphigus." The features of these two varieties of bullous disease, in themselves probably quite distinct, are curiously combined in the two patients, mother and child, whom you now see. The child is æt. 3½, and ever since her birth she has been subject to bullæ, which occur on the feet and come every summer in the hot weather, so far resembling the disease cheiropompholyx. But cheiropompholyx, as far as I know, does not exist from birth, and neither does it run in families; whereas the disease in this child has existed from birth, and it runs in the family, for the mother has also been the subject of it from infancy, and the mother's father appears to have had at one period of life a bullous eruption of a kind. So here is a bullous disease, congenital and hereditary like epidermolysis bullosa, yet having the peculiar localisation and the peculiar seasonal prevalence of cheiropompholyx. The child's health seems to be good. She does not have the disease on the hands, but the mother does sometimes. Here on the mother's foot you see a bulla full of serum just like one on her child's foot; here you see another which contains blood. What causes operate to produce them, beyond the weather, is a little doubtful. The mother sometimes thinks walking brings the rash out on her feet, and that washing causes it on her hands. The mother, you may notice, has had some lesions on the neck, which she attributes to the rubbing of her collar. That reminds us again of the resemblance to epidermolysis bullosa, of which disease I showed you an example last lecture. These cases were exhibited before the Dermatological Society of London, as I wished to get an opinion as to how I ought to classify the disease; but the verdict was not unanimous.

As to treatment, I gave the child some arsenic, but it seemed to disagree, so I reverted to local treatment, dusting between her toes a powder of boracic acid and zinc and starch. However, simple borax treatment seems to suit her best; the mother says it stops the irritation with which the bullæ are associated. Most hereditary disease is unsatisfactory to treat; it implies some deeply ingrained fault of the tissues which medicines do not easily influence.

Now here is a boy with a different sort of disease. It may be fairly called a case of "prurigo." You will notice that he is covered with the remains of small pustules, which now mainly take the form of scabs. When the disease is at its worst, all these reddened scabby places are prominent pustules. You notice also that the skin is very thick and rough for a child. The distribution of the rash is mainly on the buttocks, the trunk, the arms, and the front of the thighs. There is some on the neck; none, at present, on the face. Notice also the marked enlargement of the inguinal glands. Though at present I see no definite scratch-marks, yet the eruption is extremely irritating, and the patient is constantly scratching himself. This condition has existed for a considerable time. He is now nine

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years of age, and it began when he was three. When he first came, in the spring of last year, his skin showed some small white papules; it was also roughened and thickened, especially on the extensor aspect of the limbs; there was much scabbing, and many scratch-marks. The face and scalp were then also affected. He was subsequently taken into Colston Ward, under the care of Dr. Hensley, and there got quite well. He was sent to Swanley convalescent, but after returning home he relapsed into the condition which you now see. Probably he is developing the condition known as "Hebra's prurigo." That is a disease which begins in early life with extreme itching and (it is said) an eruption of small white papules, mainly on the extensor aspect of the limbs and on the trunk. Afterwards pustules develop (perhaps from infection by scratching), and the thickened, coarse condition of skin, and the enlargement of the inguinal glands. Hebra's prurigo was at one time thought to be limited to the Continent, but there is no doubt that it exists in England; indeed, we had about two years ago, in Matthew Ward, a well-marked case. It is generally thought to be incurable. Still, this may not be so in its early stages, and particularly if, as has been suggested, its early stage is identical with the disease we know as lichen urticatus. "Lichen urticatus" starts in children as an irritable eruption very like urticaria; but it is more persistent than a simple urticaria. Moreover the red urticarial patches often are surmounted by a little papule or vesicle. This gets scratched off, and being inoculated by the finger-nails, the rash becomes pustular, till at length a chronic condition is produced like what you see in this boy. Inasmuch as this boy's rash was cured only a year ago, it would be rather risky to say that he is as yet incurable; but he is not doing so well with out-patient treatment as he did in the hospital. At present he is having arsenic by the mouth, and inunction at night, with a solution of thirty grains of menthol in six ounces of olive oil. That appears to relieve the itching to a certain extent. I think we must next try the effect of a warm, weak sulphur bath every night, followed by the inunction of menthol or of liquid vaseline. In Colston Ward he got well without any special treatment of the skin; no doubt the rest, and the equable temperature of bed, and proper feeding, had a good effect.

The next case is that of a young man suffering from lupus. In his case the disease is peculiarly extensive. Lupus generally affects exposed parts such as the face. But here, in addition to the face, it affects the arms and the body very widely. Still, though very general, it has shown little tendency to ulcerate; so that it is a disfigurement rather than a danger to him. He is twenty-eight years of age, and the disease began when he was three.

It began on the face; you see how widely the face has been attacked. The lower eyelid on one side is everted from scarring of the face below. The lobe of one ear has been destroyed; that on the other is adherent from scarring. These raised reddish lines mark the places where scraping was done; it stopped the complaint for a time, but it began again. These patches on the body are partly fresh areas of disease, and partly scars where the disease once was. At the edges, where it still spreads, you may see the brownish-yellow nodules which are the characteristic lesions of lupus. These consist of tiny patches of tubercle, situated in the corium. It is the destruction of the corium which leads to the formation of the scars. But this large circular scar on the upper abdomen is partly due to treatment. The X rays were applied to a large patch of disease here, and their effect was rather pronounced, and produced ulceration which it was difficult to heal. The lupus patch seemed at the time to have been cured, but I regret to see that the disease has begun again around the edge, for here are little yellow-brown islets of lupus tissue still appearing. The patient himself thinks that painting the face with Donovan's solution is beneficial, and at the same time he is taking cod-liver oil and thyroid gland.

The next case, the boy whom you see, is akin to the last, though in some respects different. He had been attending here under Mr. D'Arcy Power for obstruction of the nose, I believe, the nature of which I do not know. He was sent to the skin department on March 17th, on account of a patch of disease on the skin of his nose, partly consisting of scar tissue, partly raised, reddened, and infiltrated; this was said to have ulcerated last year, and there were divers spots on other parts of the face, some reddened and some scarred. Lupus was suspected (the patient's age was that at which

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lupus might begin), *plus* a superadded impetigo, since there were gummy yellow crusts on the surface suggesting impetigo. But soon afterwards the boy developed under his chin a large, soft, infiltrated patch, in which an abscess formed, which broke and discharged, resulting in a large oval ulcer with undermined edges and a rather dirty-looking base. It became evident that we had to do with a tubercular disease of the skin, "strumous" or "scrofulous" disease, as it used to be called. It is rather difficult pathologically to draw a definite line between such tubercular disease of the skin and lupus, because lupus is a form of tubercular disease. Clinically, however, there is this difference—that lupus is a primary tuberculosis of the skin (it is the exception in a patient suffering from it to find manifestations of tubercle elsewhere), whereas tubercular infiltration and ulceration of the skin, such as we have in this boy, is generally secondary to disease of structures lying underneath it, such as bones or glands. It is not obvious, however, where the disease began in this case, unless it be the bones of the nose. Again, we do not find in this form of tuberculide the yellow-brown nodules in the corium which constitute the early stage of lupus. He has been treated internally with cod-liver oil, and locally with iodoform. I had a boy in the hospital last year suffering from tubercular ulceration of the hands (also supposed at one time to be lupus), for whom, at Mr. Lockwood's advice, we used an iodoform dressing with very good results. The method was to powder the ulcers with iodoform, and then wrap up the hand in mercurial gauze for twenty-four hours; then, after removing the dressings, sop up any discharge and scab with a little cotton wool soaked in carbolic, and re-apply the iodoform and gauze. With certain modifications I applied that treatment to this boy's chin, and what was a big unwholesome ulcer is healing up very satisfactorily indeed. It is remarkable how many of the cases of lupus and tubercular skin disease that I have seen lately in this department have been country folk. This one would hardly expect.

By way of complement and contrast to these cases, I had intended to show a case of syphilitic ulceration of the face. The patches of infiltration and ulceration which occur, often upon the face, in the later stages of syphilis are sometimes confounded with lupus. But I regret to see that the patient has not come. Instead, I show you two women with syphilitic eruptions of an earlier stage. The one has a common form of rash, partly macular, partly consisting of largish papules, more or less scaly, situated on the flexor surface of the forearms, *plus* an indefinite blotchy rash on the legs.

The other has had a profuse rash of small firm papules, very marked on the face, arms, and legs, which is now dying off but leaving a good deal of pigmentation behind. But into the very large question of syphilides in general I have not time to enter now.

Experimental Cancer Research.—Jensen has continued his studies of malignant tumours on white mice, and now announces that he has succeeded in transmitting through twenty-two generations a tumour growth originally derived from cells of a melano-sarcoma on a horse. He gives photogravures of one of the mice showing the external aspect and the dried specimen of one of the tumours, which swelled the animal to nearly twice the normal circumference of the body back of the front legs. He also gives fourteen cuts of the histologic findings. They indicate a pronounced carcinomatous structure, but there was no evidence of metastasis. The tumours continued to grow until the animals died from cachexia or from ulceration of the testicles. The tumours were transmitted from one generation to another by transplantation, and the transplantation was successful in 40 to 50 per cent. of all the attempts on white mice, but failed on other animals except on a few ordinary mice. There was not the slightest evidence in favour of a parasitic action.—*Hospitalstidende*, xlvi.

Treatment of Lupus with Refrigeration.—Sorensen reports that he has treated six cases of lupus to date with a freezing application, spraying the parts with ethyl chlorid, as practised by Detlefsen. Such remarkable improvement was obtained that he thinks we can rank the Detlefsen with the Finsen method of treating lupus as a notable progress. He regards the Finsen as the standard method, but in cases in which a course of Finsen treatment is impracticable, the freezing method enters on its rights. It is peculiarly adapted for private practice. The sittings are daily at first, then with longer intervals.—*Journ. A. M. A.*, August 29th.

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THREE LECTURES ON ACUTE APPENDICITIS.

LECTURE II.

APPENDICULAR ABSCESSES.

Delivered at the Polyclinic on February 27th, 1903.

By HAROLD L. BARNARD, M.S., F.R.C.S.,
Assistant Surgeon to the London Hospital and to the Metropolitan Hospital.

GENTLEMEN,—I will begin my lecture to-day by relating to you a case of appendicitis which illustrates many of the points to which I drew attention in my last lecture, and some to which I shall refer to-day. A young man æt. 24 rushed away one Saturday afternoon last autumn from his office in the City to catch the train from Waterloo Station to Shanklin, in the Isle of Wight. He had no time to get his lunch, and the journey was an extremely cold and inclement one. He arrived in Shanklin exhausted, chilled, and starving. He then ate a very large and apparently indigestible meal. Next morning he was seized with vomiting, pyrexia, and considerable pain in the upper part of the abdomen. On the second day he was jaundiced, and on the third day his temperature was down and he was rapidly improving in condition, when suddenly he was seized with severe pain at the umbilicus, and became collapsed. His face, I am informed, was an ashy colour. His medical man, Dr. Cowper, of Shanklin, with courage equal to his surgical skill, would not take the responsibility of allowing so acute a case of appendicitis to await the arrival of a surgeon from London, and he accordingly removed the appendix about six hours after the onset of the attack. He found a calculus, and the appendix was flaccid, green, and gangrenous. There were no adhesions, and apparently general peritonitis was present. I saw the patient next day in consultation with Dr. Gilbart Smith. The abdomen was tender all over, but not distended. A considerable quantity of free fluid was located in each flank, and another large collection in the pelvis. Three days later the general peritonitis had subsided from all the upper part of the abdomen. The fluid had been absorbed with the exception of a collection in the pelvis, which formed a soft, fluctuating mass in the recto-vesical pouch. A week later Dr. Cowper asked me to go down to Shanklin, as the collection of fluid in the pelvis had formed a pelvic abscess. On my arrival I found that it had just ruptured into the rectum. The patient made a good recovery. This case illustrates the way in which indiscretions in diet may increase the virulence of the *Bacillus coli* in the alimentary canal, and how this micro-organism attacks the appendix weakened by the presence of a calculus. It is also a good example of sudden gangrene of the appendix leading to fulminating appendicitis, and illustrates how these cases can only be saved by immediate operation. The free fluid mentioned in the above case is antibacterial in action and preserves the peritoneum from invasion by the micro-organism. The pelvic abscess was due to the whole of this fluid not being absorbed, and the rupture into the rectum I will point out later on is the most favourable termination of this form of appendix abscess.

Appendix abscesses form in two ways: either adhesions are formed before the suppurative process extends from the appendix, or suppuration breaks into an open peritoneal cavity and leads to a great outpouring of lymph into the peritoneal pouches, some at least of which may subsequently form abscesses. We will take these in turn.

When suppuration starts inside the appendix its peritoneal covering becomes inflamed, lymph is deposited on it, and the neighbouring coils of small intestine become matted together around it. If, after this has occurred, the suppuration spreads through the walls of the appendix, it finds itself shut in and circumscribed by a barrier of adherent coils of gut. In the great majority of such cases the appendix is actually perforated and its interior in continuity with the abscess formed. This is, however, not necessarily the case, and the micro-organisms may pass through the wall of the appendix and form an abscess around it without any actual perforation. It is these latter cases which supply the small minority of appendix abscesses which relapse after they have been drained and healed.

In the second class of appendix abscess, either because the appendix ruptures or the micro-organism is of such virulence that it rapidly passes through the wall of the appendix, there is no time for the formation of any adhesions, and the micro-organism enters an open peritoneal cavity. The first measure of defence is the outpouring of a large quantity of serum into the peritoneal cavity, which stands in ponds in the peritoneal pouches, to whose anatomy we must

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refer immediately. This serum is antibacterial in action, and serves to arrest, or at least to delay, the invasion of the entire cavity by the micro-organism. Should it be successful in this endeavour, the area of infection is circumscribed by adhesions and the serum is absorbed from the rest of the peritoneal cavity. Should the infection, however, be too virulent, leucocytes pass in vast number into these ponds and add their antibacterial action to that of the serum, and in this way convert them into cavities filled with pus. The leucocytes, moreover, block up the stomata of the serous membrane, and, as masses of lymph, wall in and circumscribe the septic area, so that even the toxin which the micro-organism succeeds in forming in the abscess is not absorbed into the general system to poison it. We must now turn for a moment to the clinical side of the question, and consider *the signs and symptoms which will inform us in any case of appendicitis when suppuration has commenced.*

It has been well said by Dr. Hawkins, of St. Thomas's Hospital, that no man can foretell in the first forty-eight hours of an attack of appendicitis whether it will terminate in suppuration or not. The mildest attack, attended by a low temperature and only a slight pain or two, may yet end in a large abscess, whilst another case, in which there were severe symptoms and high pyrexia, may yet resolve in three or four days without suppuration having occurred. It is, however, certain that severe attacks more often terminate in pus than slight ones. To quote again from Dr. Hawkins, the indication that suppuration has occurred is not the appearance of any new sign, but a continuance or increase of symptoms which should be abating on the fifth or sixth day. We should strongly suspect that pus has commenced to form if after the fifth day of appendicitis the temperature has not reached normal or is remaining high, or after having come down has risen again. The pulse is also a valuable indication if it remains rapid, but I have often seen a well-circumscribed abscess with a pulse-rate of not more than 84. Diarrhœa is a valuable sign; but perhaps the creamy tongue, which has not commenced to clean, and the pale, pasty, and muddy complexion of the patient are the most suspicious symptoms. This aspect of the patient is very striking in some cases, and might almost be called a purulent complexion. After the abscess has been efficiently opened and drained it almost immediately changes to a clear and rosy countenance. We may, perhaps, connect this change of appearance with the great alteration in the number of leucocytes circulating in the blood which accompanies it, for whilst the abscess is undrained there are from twenty-three to thirty thousand leucocytes per cubic millimetre in the blood, and with the draining of the abscess this number falls to eight or ten thousand. As we saw in the first lecture, abscess is much more common in the first attack. Of the twenty-seven cases in which I made a special note of the attack, the abscess formed in twenty during the first attack, in four during the second, and in only three afterwards; and in twenty-nine cases in which I noted the day on which the abscess was discovered, in twenty-three it was after the sixth day, and in only six cases were abscesses found on the fourth and fifth day, and in these considerable general peritonitis was present.

When on these general grounds we suspect that pus is present, we must discover and watch the tumour formed by the inflamed appendix with the greatest attention. It is very bad practice to await the appearance of redness, œdema, or fluctuation; one might as well await their appearance in empyemata. For many abscesses will have ruptured into the general peritoneal cavity before they produce these signs in the skin. We therefore diagnose the existence of an abscess if the lump in the abdomen continues unchanged, or especially if it increases in size after the fifth day.

The position of appendix abscesses is determined partly by the position in which the appendix lies and partly by the peritoneal fossæ in its neighbourhood which the effusion of serum may occupy. The appendix may either lie inside or outside the cæcum and colon, and if it lies inside the cæcum its tail may be either in front or behind the mesentery of the small intestine. We may therefore classify the positions of the appendix as follows:

A. *External.*	B. *Internal.*
1. Lumbar . . . = 4 cases.	1. Iliac . . . = 10 cases behind mesentery.
2. Subhepatic . = 4 cases.	2. Promontory = 2 cases in front mesentery.
3. Subphrenic. . = 4 cases.	3. Pelvic . . = 14 cases behind mesentery.
4. Retro-cæcal . = 4 cases.	4. Poupart . . = 2 cases neither.

I have noted the position of the abscess in 44 cases distributed as above.

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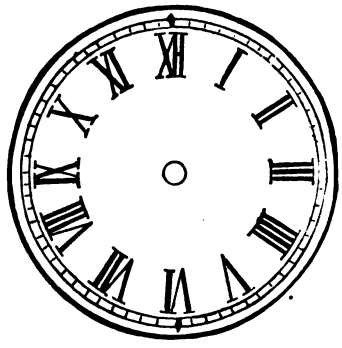
The clock mnemonic is a useful means of remembering these positions of the appendix, with their direction and significance. The appendix is compared to the hand of a clock and is supposed to rotate around its attachment to the cæcum.



I o'clock.—Sacro-iliac synchondrosis—usual site, behind mesentery.
II o'clock.—Promontory—among coils of small gut—in front mesentery—a fulminating position.
III o'clock.—Crosses iliac vessels—hangs into pelvis—felt in recto-vesical pouch—behind mesentery.
VI o'clock.—Middle of Poupart—fœtal form—dangerous—points at times through rings.
XI o'clock.—External—in furrow—felt in loin—spread subphrenic, and rarely small sac.
XII o'clock.—Extra-peritoneal—retro-cæcal—psoas myositis—perinephritic—subphrenic—bladder—pelvis of kidney.

The first of these, the iliac position, is the commonest. The appendix coming off from the posterior and inner part of the cæcum runs upwards and inwards behind the last piece of the ileum, and is shut in anteriorly by the end of the mesentery. An abscess formed in this situation is therefore well cut off from the general peritoneal cavity. As it extends the pus comes forward in the angle between the ileum and the cæcum, and reaches the anterior abdominal wall in this situation. Clinically we find the tumour situated under McBurney's point, with an interval between it and the anterior superior spine and Poupart's ligament.

In the second of the internal positions the appendix passes from its origin upwards and inwards toward the promontory, but passes in front of the mesentery and behind the omentum. Here it lies in the centre of the abdomen and among all the coils of small intestine, with no part of the abdominal wall anywhere near it. I consider this position to be a most dangerous one, and in the only two cases in which I have definitely located the appendix in this situation it has led to fulminating peritonitis.

In its third position the appendix passes directly inwards across the iliac vessels and beneath the mesentery, and hangs dependent into the recto-vesical pouch in the male, or Douglas's pouch in the female. We shall consider this pelvic abscess in detail later. But it may be said at once that the abscess formed by an appendix hanging into the pelvis can only be reached by a finger in the rectum or vagina.

In its fourth situation the appendix passes directly downwards and lies upon Poupart's ligament in the inguinal furrow. This always occurs in the fœtal form in which the appendix arises from the extremity of the cæcum. It is in my opinion a dangerous form of appendix abscess, for lying as it does at the edge of the mesentery, the pus may either pass in front or behind that structure, and should it escape between the omentum and the mesentery it will reach the central part of the abdomen occupied by the numerous coils of small intestine which are most sensitive to infection and produce the greatest shock. This abscess can easily be felt as an indurated mass lying above the outer half of Poupart's ligament. It sometimes escapes by the inguinal canal and the external ring.

In Fig. 1 are illustrated the peritoneal pouches, which will be invaded by an abscess originating from an appendix which lies between the ascending colon and the right loin. Such an appendix may be long enough to reach the lower border of the liver, and lie either outside it beneath the diaphragm or, passing below its inferior surface, may touch the gall-bladder. But it is not necessary for an appendix to actually reach these situations in order to produce abscesses there. In this figure, No. 1 represents a lumbar abscess. As you will see, it is shut in between the ascending colon and the loin, and the twisted mesentery of the appendix serves to prevent pus passing downwards. Clinically it produces a tender, resistant tumour, passing back along the iliac crest from the upper part of the iliac fossa, and extending up the loin both laterally and behind. Indeed, the best sign of an appendix in this situation is the extreme tenderness on deep palpation at the outer border of the erector spinæ. If the pus extends up to the lower border of the liver it readily passes below it to the left, being bounded below by the transverse colon,

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above by the liver, and behind covering the upper two-thirds of the right kidney (Fig. 1). Such an abscess produces a large area of dulness and induration on the right side of the abdomen in front, continuing downwards from the right costal margin and the lower border of the liver. The peritoneal fossa in which this abscess

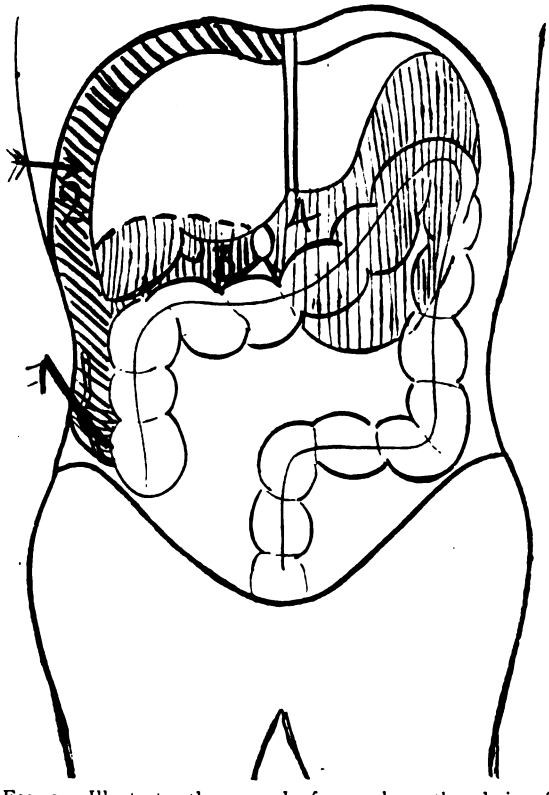

FIG. 1.—Illustrates the spread of pus along the chain of right peritoneal ponds from an external appendix abscess 1. The appendix in external position lying in a lumbar abscess. 2. Subphrenic intra-peritoneal abscess between liver and diaphragm, and limited to the left by the falciform ligament. 3. Subhepatic abscess between the lower surface of liver and the upper end of right kidney. 4. Small sac of peritoneum very rarely invaded through the foramen of Winslow from 3. The oblique loin incision is represented; through it tubes may be passed into 2 and 3. When 2 is shut off and circumscribed it must be opened across the pleura in the line of the arrow.

forms is the same one into which the bile tends to drain after operations on the gall-bladder and bile-ducts, and Mr. Rutherford Morison, of Newcastle, has fully described its anatomy in this connection, and has recommended that it should be drained in these biliary cases from the loin. In a few rare cases the suppuration has extended still further to the left from this subhepatic fossa by passing through the foramen of Winslow and distending the sac of the small omentum, as is shown in Fig. 1 (4).

To turn back to the point at which the pus reached the lower border of the liver, it may as well as passing below the liver pass between that organ and the diaphragm, and form a large subphrenic abscess in the situation numbered 2 in Fig. 1.

We shall refer to the clinical signs by which this subphrenic abscess may be recognised when we come to deal with these abscesses in detail. The last position of the appendix is one in which it passes directly up behind the cæcum and ascending colon, and lies apparently outside the peritoneal cavity altogether, in relation with the psoas muscle, the right kidney, and the cellular tissue around these structures. But this abscess also will be dealt with in detail later.

I will now describe to you *the principles which should guide us in opening any of these abscesses*, with the exception of the pelvic, subphrenic, and retro-cæcal abscesses, which will require special treatment. The situation and limits of the mass formed by the abscess should be most carefully defined, and we should then so plan our incision through the anterior abdominal wall that it may be as far removed as possible from the general peritoneal cavity. We should, indeed, approach the abscess from the outer side, in order that as little risk as possible may be run of infecting the general peritoneum by allowing the pus to pour into it as we release it from the abscess. No formal incision is suitable in every case, and you will see by the diagrams I show you the deplorable results of making orthodox incisions where they are not applicable to the abscess which is being opened. For these figures I am indebted to Mr. James Berry. He drew them from actual post-mortems made by him at St. Bartholomew's Hospital. The adhesions formed by the abscess to the anterior abdominal wall are marked in each case, and you will see that in scarcely any instance was the incision made within this region. In nearly every case the surgeon incised either in the middle line below the umbilicus and entered the abscess cavity from the general peritoneal cavity, infecting it by so doing, or his incision was the orthodox oblique one opposite the anterior superior

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spine, and in a similar way the pus was allowed to run from the abscess into the open peritoneal cavity.

As layer after layer of the abdominal wall is cut through we must notice the presence or absence of œdema between these layers as we approach the peritoneum. If well-marked œdema is present we can be certain that the abscess has formed adhesions to the abdominal wall, and whilst there is little risk of infecting the general peritoneal cavity, we must avoid wounding the adherent coils of gut. Should open peritoneum be encountered, we must pack it off with a barrier of gauze all round before proceeding any further. For this purpose it is usual to employ broad iodoform gauze with a woven edge, and to systematically pack it in around the incision, starting at one end from the point where the abscess wall is adherent to the abdominal wall, and working systematically round the wound to the point on the other side where it is similarly adherent, so that there may be no crack or crevice leading from our wound into the peritoneal cavity which is not blocked by the gauze barrier. The effectiveness of this method in protecting the general peritoneal cavity from infection has often filled me with astonishment, when I have seen foul and stinking pus pouring from a large appendix abscess and beating against the narrow barrier of iodoform gauze which I have placed to prevent it flowing into the peritoneum. It seems that this gauze acts in several ways: mechanically it serves as a dam and makes it easier for the pus to flow from the wound than to percolate through the gauze; then it appears that it acts as an irritant upon the coils of gut upon its further side, and makes these adherent to one another before the micro-organisms have overcome the obstacle it offers to their advance. It is possible, moreover, that it acts as a filter, straining off the micro-organisms themselves, but permitting their toxins and poisons to soak through its substance and excite a large formation of lymph, which effectually bars the progress of the bacteria themselves when they reach it. When the gauze barrier has been prepared the index finger is employed to break down the adhesions between one coil of gut and the next and to open a way into the abscess cavity. This aperture, again, should always be made on the outer side, and as far as possible from the central region of the abdomen. There has been much dispute as to whether the appendix should be removed when an abscess is opened or not. It is claimed that some cases of appendix abscess relapse even after the abscess has been drained and healed. But these cases of relapse are very few. In Dr. Hawkins' cases one relapsed in twenty-three, and in a great number of cases collected by Coley the relapses were estimated at 20 per cent. In forty-two cases of appendix abscess of which I have notes, only one case has had recurrence, but these cases have occurred only in the last three years. We must remember, also, that the drainage of well-localised appendix abscesses is attended by a mortality of not more than about 5 per cent., but that if the appendix is sought for and removed in every case it will necessitate a large incision, with its greater risk of ventral hernia, extensive breaking down of the adhesions, which must in many cases lead to general peritonitis and a very much prolonged operation, which must take a further toll in consequence of the shock it produces. Supposing that even 20 per cent. relapsed, these may be operated on in the quiescent period with the 1 per cent. mortality to which we have so often referred. When the abscess has been opened, a thorough but gentle search should be made for fæcal concretions or calculi. For if they are left in the abscess cavity they will lead to the formation of a fistula, and unless they are ultimately disintegrated and discharged the abscess will recur. I believe that the largest drainage-tubes should always be employed for these abscesses, and I prefer them three quarters of an inch in diameter. After the second day a full-sized soft catheter attached to a syringe may be inserted to the bottom of this large drainage-tube, and the cavity of the abscess be thoroughly well washed out with 1 in 80 carbolic lotion. This washing is continued until the lotion comes back clear, and the abscess cavity is then sucked dry by reversing the action of the syringe. These big tubes should not be taken out to be cleaned during the first week, for it will be found very difficult and painful to replace them, more especially as when they reach into the pelvis they are six or seven inches long. They should, however, be turned round each day in order that the lateral openings may not become blocked.

Pelvic Abscesses.

I wish to especially draw your attention to pelvic abscess in appendicitis, because in the first place it

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is far more common than seems to be supposed; and secondly, of all appendix abscesses it is the most insidious, with the exception perhaps of the subphrenic abscess to which I shall refer later. It is a deplorable thing when a patient has safely passed through all the dangers attending the first week of an acute attack of appendicitis, and appears well nigh out of danger, that he should then succumb to a pelvic abscess which has lurked in the pelvis and developed until it has finally involved the whole peritoneal cavity and led to the patient's death at this late stage of the disease. The reason that pelvic abscess is so often overlooked is because they can only be diagnosed by examination from the rectum or vagina. It is, therefore, an absolute axiom of appendix surgery that in every case of appendix abscess the rectum must be examined. And it is a useful aphorism that if in a case supposed to be appendicitis the appendix cannot be located in the abdomen it will be found in the rectum.

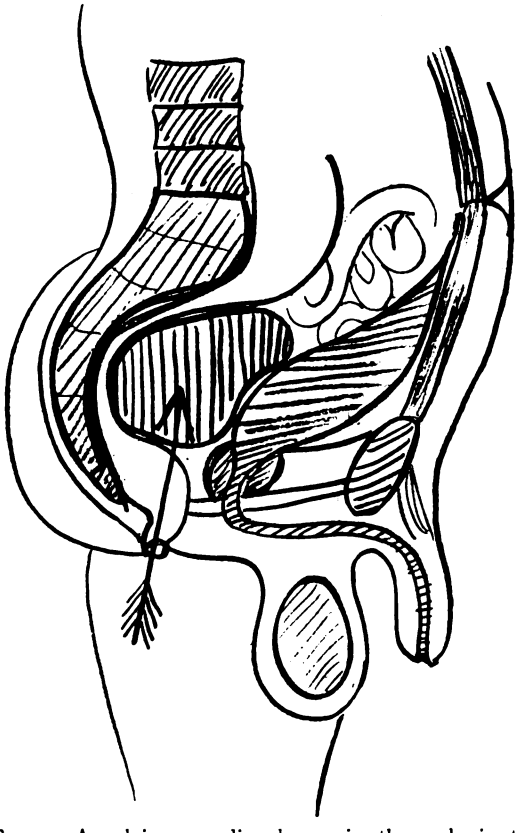

FIG. 2.—A pelvic appendix abscess in the male in the retro-vesical pouch. It is easily felt from the rectum just above the prostate as a more or less fluctuating, tender mass. It is best opened at this point, as the arrow indicates. It compresses the rectum and dilates the entire colon above it. It presses on the bladder, and more often than in the female leads to frequency or retention. Always empty the bladder with a catheter before opening such an abscess from the rectum. Matted coils of gut roof it in above.

The abdominal condition which should arouse our suspicion that an abscess is deeply seated in the pelvis is as follows:—The abdomen is generally distended and tumid, but it is not sufficiently tender or rigid for us to attribute this distension to peritonitis. This distension is indeed due to obstruction of the rectum in the pelvis by the abscess there (see Figs. 2 and 3), and, as a consequence, the entire colon, and in advanced cases the small intestine, become dilated. No definite lump can be found in the iliac fossa or elsewhere. But in advanced cases there is an obscure resistance above the pubes due to the dome formed by matted coils of gut above the abscess cavity. This obscure resistance is resonant on percussion. In nearly every instance I have, however, found a tender spot over McBurney's point. In many cases the abscess, lying as it does between the rectum and bladder, produces symptoms referable to irritation of these organs. There is diarrhœa and tenesmus, with the passage of frequent motions containing much slime, and the urine may be either frequently voided, or in rarer cases actually retained. In one case the signs and symptoms were much complicated by retention, and a bladder distended with twenty-four ounces of urine. When this had been withdrawn by a catheter a further large fluctuating mass was found behind it which proved to be a pelvic appendix abscess. In a case exhibiting such signs, if we examine by the rectum we find the condition shown in Fig. 2. Situated above the prostate and in front of the rectum is a large, tender, sometimes hard, and sometimes fluctuating mass, frequently the size of an orange. The mucous membrane of the rectum over it is often swollen, œdematous, and secreting the slime to which we have already referred. The arrow in the diagram indicates not only the point at which the examining finger will impinge upon the abscess, but also the point at which it is best opened, as we shall describe later. We also see in the figure how the rectum is flattened against the sacrum behind, and how in front the abscess compresses the neck of the bladder, whilst above is indicated the matted coils of gut which shut the abscess in and separate

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it from the anterior abdominal wall. In the female (Fig. 3) the abscess can in addition be palpated in the posterior fornix of the vagina, where Douglas's pouch will be found distended and bulging; and, as I shall point out later, this is the point of election from which to open such an abscess. It will be readily understood that an appendix hanging down into Douglas's pouch will be in close relation to the right ovary and Fallopian tube, and in its early stages, when adherent to the ovary, it may be very difficult to tell whether the pain is due to ovaritis or salpingitis, or to an inflamed appendix adherent to these structures. In some cases it is even difficult to be certain in which of these organs the mischief arose after the abscess has been opened into the vagina. But the character of the pus, which is usually characteristically foul in appendicitis, will, in most cases, correctly indicate its origin.

I have during the last three years closely investigated fourteen cases of pelvic appendix abscess which had reached different stages of development, from a localised collection of pus in the pelvis up to general peritonitis which led to death; and I have been able from this investigation to trace the course by which the abscess spreads during the latent or insidious period, and this course is shown, as it were, in a map in Fig. 4. In many cases pelvic abscesses perforate the wall of the rectum and discharge themselves as a sudden fluid motion. I have in two such cases been able to identify with my finger the point of perforation. This termination is a very favourable one, for in a fortnight all discharge has ceased, and nothing remains but some induration in the recto-vesical pouch. In other cases, however, the abscess, starting from its point of origin in the pelvis, gradually expands and lifts the firm dome of adherent coils of gut as though it were the lid of a pot, and the pus then escapes round the edges behind into the right or left iliac fossa (see Fig. 4). Since the abscess at its start is behind the mesentery, as it spreads it keeps behind the small intestine, and these pushed forward against the abdominal wall serve to conceal it as it creeps from the iliac fossæ on either side to the loin, and from the right loin below the liver and diaphragm, and on the left around the spleen. In a recent case in a boy I found large collections of pus in all these situations, and yet the temperature was not more raised than 100° Fahr., the pulse was below 100 per minute, and the abdomen exhibited nothing except diffuse distension. The deep posterior situation of the abscess as it spreads is very fortunate in one way, for the pus in its entire course skirts round the edge of the mass of intestines, and does little harm where it lies shut in against the posterior abdominal wall, but the very effectiveness of this isolation makes this stage

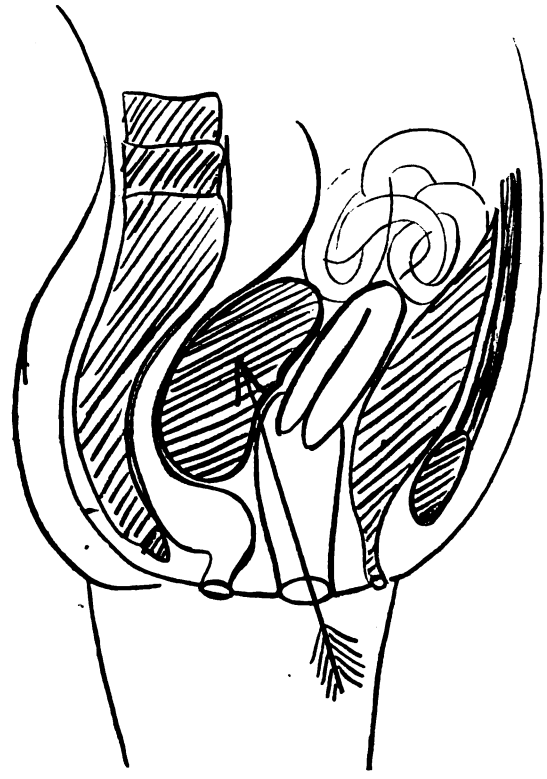

FIG. 3.—Pelvic appendix abscess in the female. It is felt from the rectum in Douglas's pouch, and obstructs the rectum, distending the whole colon. It is felt from the vagina in the posterior fornix. The arrow shows how and where it should be opened. It compresses the bladder in front, causing painful and frequent micturition, and, very rarely, retention. The dome of matted coils of gut above shut off the general peritoneal cavity.

of development insidious, and leads to its being overlooked unless it is suspected. There are two other collections of pus which may extend from those I have mentioned. The subphrenic abscesses may infect the pleura through the diaphragm and lead to local or general empyemata at the bases of the lungs. In my wards at the London Hospital at the present time is a young fellow in whom a

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pelvic abscess tracked up the left loin and formed a left subphrenic abscess. Both these collections of pus were emptied, but two days later he coughed up a large quantity of stinking pus and gas apparently from a localised empyema at the left base; and this last collection is being evacuated so well by these natural means that I do not propose to resect a rib, at least at present. At post-mortems on two of these pelvic cases which came into the hospital with general peritonitis due to the final development of a pelvic abscess, in one case fourteen days after the onset of the attack I found a further collection of pus (see Fig. 4), which was situated between the descending colon and the spine in the hollow of the left loin, roofed in by the posterior surface of the mesentery; and I have not been able to determine whether this is the usual route by which pus escaping from a pelvic abscess passes up the left loin and reaches the left subphrenic space, or whether, as on the right side, it tracks up outside the colon. In its final stage of development the subphrenic abscesses right and left rupture from above, and the pus passes down towards the central region of the abdomen. Then acute symptoms of general peritonitis at once supervene, and this is unfortunately often the first point at which the doctor becomes aware that the case is not progressing as it should. Up to the point at which this occurs the patient is still in good condition, and may be saved by suitable incisions, so planned as to drain all these collections of pus. These incisions, which are marked on the diagram, are first a medium one just above the pubes. After gauze has been packed in from one Poupart ligament to the other in the form of a rainbow, the index finger is steadily bored down the back of the bladder, perforating the dome of adherent coils of gut, until it slips into the cavity of the abscess. A large drainage-tube about six inches long is then passed down to the bottom of the pelvis. Two lateral incisions are also necessary, one in each loin. These are placed as far back as possible, and through the right one tubes are passed below the liver, under the diaphragm, and down the loin to the iliac fossa. Through a similar incision on the left the abscess around the spleen is drained above, and another tube is passed down into the left iliac fossa. A small boy of twelve to whom I have already referred was treated by these incisions two months ago. A pelvic, two iliac, two lumbar, and two subphrenic abscesses were opened, and he is now convalescing at the seaside with all his sinuses healed.

The Treatment of a Pelvic Abscess.—It is always better to drain a localised pelvic abscess from the rectum or vagina. By either of these methods the patient is well in a fortnight and never in the hospital more than three weeks. Moreover they run no risk of ventral hernia. On the other hand, if they are drained by a supra-pubic incision the wound takes six weeks at least to heal, and often much longer. It must, however, be admitted that the cases which I drained from the abdomen were more severe and extensive cases, or I should not have adopted this method. If, however, the abscess has already spread to the iliac fossæ and the loins, it is not enough to open the pelvic abscess. How, then, can we tell whether a pelvic abscess is still localised as shown in Figs. 2 and 3, or whether it has already started on the development shown in Fig. 4?

We can, guided by a knowledge of the course it will take, palpate the iliac fossæ and the loins, but, as I have already pointed out, the pus is very deeply situated and is covered by all the coils of small intestine. Some fulness can, however, usually be discovered in these situations. A more valuable sign is marked tenderness on deep pressure at the outer edge of the erector spinæ in the loin behind. The best indication of all, however, is gained by palpating the abscess cavity with a finger in the rectum. If it is hard and tense the abscess is still shut into the pelvis. Directly the pus escapes over the brim of the pelvis the abscess as felt from the rectum becomes soft and flaccid, and can with difficulty be distinguished from a distended bladder or dilated coils of gut.

Should we then have decided that the abscess is still limited to the pelvis, and the patient is a male, the abscess should be opened into the rectum (see Fig. 2).

The anus is first dilated, the sphincter paralysed, and the bladder is emptied by a catheter. The rectum is well douched. The index finger searches for a soft spot on the tense mass above the prostate, and a large exploring needle on a syringe is plunged into it. If pus is found, a sharp-pointed director follows the needle, which is then withdrawn, and sinus forceps are passed up the groove of the

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director, and by opening these a hole is torn sufficiently large to admit the index finger. A large tube is then inserted and cut short enough to rest on the upper surface of the sphincter. Rectal washes are given twice a day. The tube can be discarded after three or four days, and the patient is allowed up as soon as the discharge of pus ceases, which is usually in fourteen days.

Should the patient be a female, it is, I think, better to utilise the vagina for drainage (see Fig. 3). With curved scissors the mucous membrane and the vaginal wall are cut through in the middle line immediately behind the cervix. A director is then passed into the pus cavity, and the procedure is then exactly as in the male. The results are just as good.

When I commenced to open pelvic abscesses I had a great antipathy to doing so from the rectum, for this appeared to me a dirty procedure, and one likely to infect the abscess cavity. At this time I heard of a case drained by Mr. Goodsall through the ischio-rectal fossa, and I adopted this method in two cases. An inch incision is made into the ischio-rectal fossa by the side of the anus, and the index finger bores its way through the fat to the top of that cavity. A sharp director is then passed up the finger and impinged against the levator ani muscle which separates it from the abscess. The other index finger in the rectum serves as a guide. The director is then bored through the levator ani and recto-vesical fascia into the recto-vesical fossa, entering it from the side. Sinus forceps are passed in and the opening dilated as much as necessary. On the third occasion on which I followed this procedure the abscess burst into the rectum as the director was being bored through the dense tissues —a procedure which requires considerable force,— and this patient did perfectly well. Soon afterwards the abscess ruptured into the rectum whilst another case was being examined on the table prior to making the ischio-rectal incision. This case also did well. I then abandoned the ischio-rectal route, as it presented no advantages over the rectal method, and had, indeed, several disadvantages. The sinus took some week or so longer to heal, and a colleague who employed the method encountered fairly severe hæmorrhage. Finally, it appeared to me that if the force required to perforate the levator ani and recto-vesical fascia were enough to burst the abscess into the rectum in one case, in another it might rupture it into the general peritoneal cavity.

Should the signs which I have already enumerated of fulness in the iliac fossæ, tenderness in the loins, and a flaccid mass felt by the rectum, indicate that the pelvic abscess has already spread to the iliac fossæ, or even further, then the abscess must be opened in the middle line above the pubes. Before the abscess is opened or the adhesions disturbed its limits may be cautiously explored from this median incision and the necessity of lateral

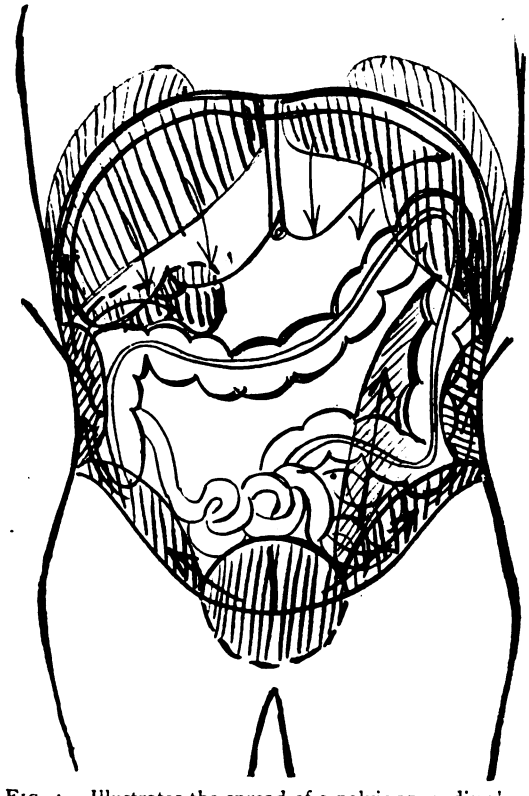

FIG. 4.—Illustrates the spread of a pelvic appendix abscess along the right and left chains of peritoneal ponds. The abscess is at first deeply localised in the pelvis (see Figs. 2 and 3). It then lifts the dome of matted gut and escapes into one or both iliac fossæ. On the right it then spreads along the pouches already illustrated in Fig. 1 to the right subphrenic space. On the left the pus similarly reaches the subphrenic space around the spleen. In some cases it passes between the descending colon and the loin; in others, between the descending colon and the spine, lying on the front of the kidney, and shut in by the back of the mesentery. Up to this stage all these abscesses may be drained by the incisions indicated in the figure—one median one, and two oblique ones far back in the loin. Finally, the subphrenic abscesses come down the front of the liver and invade the central dangerous area of the abdomen, or spread through the diaphragm, producing empyemata or pericarditis.

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loin incisions determined on. When this point has been decided the rainbow packing of gauze is inserted from one of Poupart's ligaments to the other, and the abscess opened as already described.

On one or two occasions when I had found pus in one loin and drained it, and proceeded to incise the other, I found so little pus there that it was uncertain whether it had reached the subphrenic or subhepatic spaces.

I nevertheless passed the exploring finger into these pouches, and as it was withdrawn as a rule a gush of serum more or less turbid followed it. But after these pouches have been thus exposed to contamination a tube should always be passed into them whether they were found to contain pus or not.

Sometimes whilst opening an obvious collection of pus in the right iliac fossa one suspects from its distribution or from a previous rectal examination that there is a secondary collection in the pelvis, as was the case in the patient whose history I related to you at the beginning of this lecture. The index finger should then be slipped across the iliac fossa until it passes the iliac vessels and drops into the pelvis, when serum or pus will gush out along it. A tube should then be slid down into the bottom of the pelvis. This tube will require to be six or seven inches long.

I have already described the method of draining an advanced case of combined pelvic, iliac, lumbar, and subphrenic abscesses. The incisions may be seen marked on Fig. 4.

Appendix Subphrenic Abscesses.

The history of these abscesses is nearly always as follows:—An external appendix abscess has been opened by an incision in the right side running a greater or less distance back into the loin (see Fig. 1). Although apparently the abscess is well drained, the patient does not do well. He displays those constitutional signs of appendix abscess to which we referred at the beginning of this lecture. His temperature is still raised, his pulse does not come down to normal, his tongue is creamy, his complexion pale, pasty, and muddy. Perhaps he has five or six motions a day. The abdominal examination reveals no further collection of pus. The pelvis is clear. Where, then, is the pus? Under these circumstances we must repeatedly examine the base of his right lung for signs of pus hidden beneath the dome of the diaphragm.

If the original abscess was a pelvic one the bases of both lungs must be most carefully watched (see Fig. 4). If there be an abscess the line of liver or splenic dulness will be found to slowly rise towards the angle of the scapula. A sudden increase of dulness in this region indicates that the pleura, in anticipation of invasion from below the diaphragm, has poured out a copious flood of antibacterial serum. The stethoscope will in some cases reveal the tubular breathing of compressed lung, in others a harsh pleural rub or, where pleural effusion has occurred, the absence of breath-sounds. As soon as the signs justify it a large exploring needle on a syringe should be inserted. I usually do this in the line of the angle of the scapula, and between the tenth and eleventh ribs; and if no pus is discovered at a depth of three inches I explore in a similar way the ninth, eighth, and seventh spaces. When the needle enters the liver dark venous blood is withdrawn. When the blood is withdrawn from the lung it is bright and scarlet, and often contains air-bubbles or even foams. Not only in this way can one tell that the point of the needle is through the diaphragm, but if this is the case at each inspiration the syringe is tilted upwards as the needle is carried down by the diaphragm. It is possible to withdraw pus from below the diaphragm and clear serum from above it in certain cases.

I have here a patient who exhibited this sign. He had an external appendix abscess which was opened and drained through an oblique incision in the right loin, the scar of which you can see. Subsequently he exhibited the signs and symptoms of a further collection of pus, the situation of which was indicated by a rapid rise in dulness from his liver to his interscapular region on the right side.

Under an anæsthetic a needle was inserted into his ninth intercostal space in the line of the angle of his scapula; a syringeful of foul pus was immediately withdrawn.

Two inches of his ninth rib were resected subperiosteally. Curved needles were then passed so as to suture his diaphragm to his intercostal muscles, and in this way shut off his pleural cavity before it was opened. An incision was then made through the diaphragm, and about half a pint of

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foul pus and gas released from under its dome. The question then arose as to what was the cause of the dulness so high in the thorax; another needle was accordingly inserted between the fifth and sixth ribs in the interscapular region, and clear serum was withdrawn. Dr. Bulloch, bacteriologist to the hospital, reported on both specimens. The pus contained *Bacillus coli communis* and streptococci. The serum was sterile.

In this other patient the history was almost identical, but the subphrenic abscess was sooner discovered, and was smaller and more restricted to the top of the dome of the diaphragm. It was accordingly the eighth space in which the needle struck pus, and the eighth rib was resected.

In yet another case in a young Jewess, when the ninth rib was resected the diaphragmatic and parietal pleura were found adherent to one another by lymph so that they could not be identified. I unfortunately omitted to insert sutures fixing the diaphragm to the intercostals. For two days she did well; then, as the abscess beneath it emptied, the diaphragm descended, and in the middle of the night it tore away from its attachment to the chest wall. Air freely entered the right pleura and the lung collapsed. The respirations rose to 54, and the pulse to 150. However, she rallied, and the lung re-expanded in due course. Ultimately she made a good recovery. These sutures should therefore never be omitted whether the diaphragm is adherent to the chest wall at the time of operation or not.

As I have already stated, if a collection of serum or pus is suspected at the time an appendix abscess is opened in the loin, the index finger is slipped up to the edge of the liver and is inserted below it, and between it and the diaphragm. Tubes are then introduced whether pus is found or not.

Retro-cæcal Appendix Abscess.

An appendix lying behind the cæcum and ascending colon, and outside the peritoneal cavity altogether, is anatomically unknown.

Pathologically, however, we frequently find an appendix in this situation, and from its extra-peritoneal position it may produce most anomalous signs.

Fig. 5 shows you the two ways in which this condition may be brought about.

More commonly the appendix was originally an external one (Fig. 5). It was well tucked down into an unusually deep groove between the ascending colon and the loin. When it became inflamed dense adhesions were formed outside it between the ascending colon and the parietal peritoneum, and as these became converted into fibrous tissue the appendix came to lie directly behind the colon and practically outside the peritoneal cavity altogether. I have seen several stages of this process of exclusion from the peritoneal cavity.

What I believe to be a rarer origin of a retro-

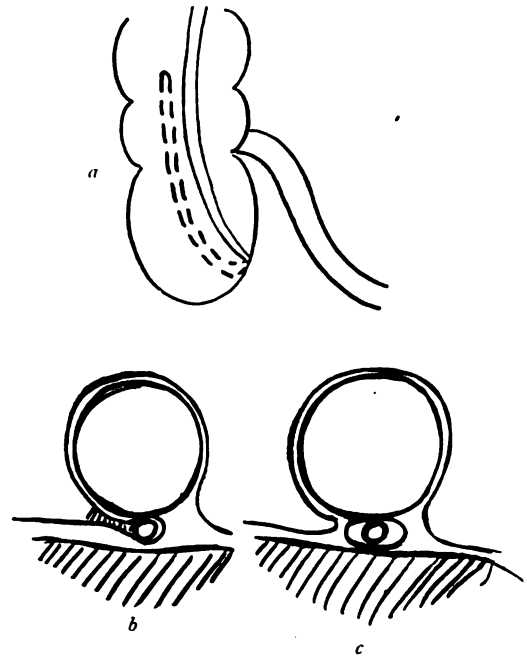

FIG. 5.—Illustrates the manner in which the appendix becomes retro-cæcal and (*a*) the position of the retro-cæcal appendix. (*b*) The first method is by an external appendix lying in a deep sulcus between the colon and the loin, being shut in by adhesions between these two structures. (*c*) The second method is by the hernia of an appendix into a retro-cæcal pouch, the mouth of which is closed by adhesions.

cæcal appendix is when it is placed in an unusually deep retro-cæcal pouch of peritoneum (Fig. 5, *c*).

In whichever manner it reaches this extra-peritoneal position, it lies in consequence of it in very close relation with the right psoas muscle, the right kidney, and ureter, and the cellular tissues around these structures. In some cases the signs when such an appendix becomes inflamed, and especially when it suppurates, are chiefly referable to the psoas muscle.

In such a case which I saw under the care of a

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colleague, the hip was flexed at a right angle, and the boy would allow it on no account to be extended. His temperature was high, but he had vomited very little. Owing to its deep position the signs were very obscure from the abdomen, but an attempt was made to reach the pus through the peritoneum. This was only in part successful, as the suppuration was already inside the psoas sheath. Subsequently the psoas muscle was cut down upon below Poupart's ligament, and sinus forceps were passed up inside the sheath. A considerable abscess was thus opened, and the boy ultimately did well.

The close relation of an appendix abscess in this situation to the kidney, ureter, and cellular tissue of the loin is well illustrated by this patient.

He has, as you see, what appears to be a kidney incision in his right loin. When he came to my out-patients' in the first instance he had a tender, fluctuating swelling in his right loin below the tip of the twelfth rib. He asserted that when the swelling was there he was in pain and felt ill, but that it disappeared at intervals, and he then felt better, but his urine became thick and cloudy.

The swelling was undoubtedly an abscess pointing through Petit's triangle, and his urine, although apparently clear, showed pus-corpuscles under the microscope.

He was taken into the hospital and his account confirmed. Subsequently I cut down on the abscess and followed it through a hole in the lumbar fascia into the retro-peritoneal tissues, but it then turned downward into the iliac fossa. I explored the kidney and the ureter for at least three inches, but could find no communication with the abscess cavity. The latter was scraped out thoroughly, except one pocket which ran down over the crest of the ilium.

The pus entirely disappeared from the urine and did not recur. The origin of the abscess from the appendix was confirmed by the formation of a fæcal fistula some days after the operation. On one or two occasions enema fluid escaped from the wound. Before he left the hospital he had what was apparently an attack of appendicitis, the resistance and tenderness being far back in the loin. This attack cleared up rapidly when his bowels were opened. He still has a sinus, as you see, and the amount of pus discharged by this varies considerably; but as he can work well and it does not inconvenience him, he does not wish anything further done. As the pus did not enter the pelvis of the kidney or the ureter we must suppose it had tracked down the latter and that the abscess opened into the bladder.

In some instances the pus of a retro-cæcal appendix may form a perinephritic abscess, or even extend upwards behind the peritoneum until it reaches the non-peritoneal surface of the diaphragm, where between the layers of the coronary ligament of the liver it will form a subphrenic abscess which is extra-peritoneal.

The treatment of the retro-cæcal form of appendix abscess, as you will perceive, differs in each case. Some are best approached across the peritoneum by an incision running back towards the loin; the adhesions between the colon and the parietal peritoneum are then broken down and the abscess drained.

Some require the incision below Poupart's ligament, as already described, and others a posterior retro-peritoneal incision, as in the boy I have shown you. If a subphrenic abscess forms it is drained across the pleura in just the same manner as though it were the more common intra-peritoneal form.

We have received from Messrs. Burroughs Wellcome and Co. a sample of a useful cardiac stimulant and chalybeate tonic made by combining iron, arsenic, and digitalin. The preparation is issued in tabloid form and its exact composition is as follows:—Soluble phosphate of iron, gr. 3; arsenious acid, gr. $\frac{1}{100}$; and digitalin, gr. $\frac{1}{100}$. A point of some practical importance in this tabloid is that the soluble phosphate of iron employed possesses the advantages of solubility and freedom from any tendency to cause constipation. We have also received specimens of "Tabloid" iron phosphate and iron hypophosphite. The favourable results which have followed the use of iron hypophosphite in phthisis, as well as in nervous affections, have been widely observed, and this salt is now prescribed with increasing frequency. This tabloid has the following formula:—Soluble phosphate of iron, gr. 2 (0·13 gm.); soluble hypophosphite of iron, gr. 1 (0·065 gm.). This combination has been found of service in cases of nervous depression associated with anæmia, and also under certain circumstances in phthisis.

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A LECTURE ON AUTO-INTOXICATION.*

By ALLAN MACFADYEN, M.D., B.Sc.

GENTLEMEN,—In choosing auto-intoxication as the subject of this lecture I have not been led to do so on account of any experience I possess as a clinical observer. The work in which I am mainly engaged is in the field of experimental medicine—in the study of the nature, origin, and possible prevention of the processes of disease—rather than in the study of clinical symptoms and the methods to be adopted for their treatment. In this latter respect each member of this audience will be much more competent to speak than I am. At the same time, whilst certain questions can only be adequately considered with the aid of clinical experience, there are others for which the proper field of investigation is the laboratory. One may even go further, and say that the advance of medicine in the future will be largely dependent on the progress made in the experimental and research side. This is widely recognised at the present time—the clinical laboratory forming an essential adjunct to every important hospital, and the student given the opportunity of appreciating the aid that is to be obtained by a diligent use of the microscope and the test-tube. The theoretical and practical aspects of health and disease problems must mutually influence each other. The more therefore that observers, whether at the bedside or in the laboratory, come into contact, the better it will be for them and for the advancement of their common cause—the relief of suffering humanity. And it appears to me that there is nothing more calculated to counteract the deadening influences which are apt at times to make themselves felt in our professional work than the endeavour to understand and

* Delivered at the Medical Graduates' College and Polyclinic.

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further the scientific aspects of medicine, even if this does not for the moment appear to bear directly on treatment. In this respect every medical man can be an observer, and contribute something to the knowledge and advance of his subject. Robert Koch laid the foundation of his subsequent fame whilst filling the position of a humble country doctor, and there are many other instances. It is a great advantage to have a school like this, upholding the doctrine that we must always be students. The success of the greatest leaders of medical thought has been in no small degree due to the experimental work they were able in the first instance to accomplish. The work at present being accomplished in physiological and pathological research has in many respects a direct bearing on the preservation or restoration of function. The subject, for example, which I have chosen — auto-intoxication — has been not only clinically, but notably experimentally, a matter of investigation. Any accurate data obtained have been mainly the result of experimental methods, and the recent advances in physiological chemistry form the basis of any accurate knowledge we possess. Work in this direction is still in its infancy, and its development, on account of the inherent difficulties to be overcome, will necessarily be slow.

We may most usefully begin by considering some terms in common use, and in this way endeavour to define our subject and its scope. It is not in this instance an easy matter, as the terms are apt to be used loosely, and their significance to fluctuate from time to time. In considering the origin of disease processes, we have to bear in mind the agents which come *from without* and those which come *from within*. As regards the morbid agents in question, we are usually in the habit of speaking of infections and intoxications. At the same time it is difficult to hold these two terms apart, inasmuch as all the symptoms resulting from an infection may be those of an intoxication; whilst a disease having every appearance of a pure intoxication may yet have its roots in the action of some infective agent and its products. The typical infective agents from without are, of course, the micro-organisms in so far as the individual species are capable of self-multiplication in the human body, and in this way leading to diseases of parasitic origin, which may culminate in a fatal intoxication of the system. At the same time an intoxication from without may occur independently of living agents, as, *e.g.*, through noxious gases and various chemical substances. Further, the intoxication that occurs, although unaccompanied by living agents, may yet be due to a poison elaborated by some such agent, which remains itself external. We have the best analogy in the case of the yeast-cell and the alcohol produced by it. The various decompositions which occur in foodstuffs before consumption may also give rise to toxins which affect the system *per se*, but which have their origin in the activity of micro-organisms. The large group of food-poisons belong to this category. And if we consider the undoubtedly parasitic forms of life, we find that in many instances the infection is quite a secondary factor, and it may be of little significance in itself were it not for the poisons elaborated at the seat of invasion, which determine the grave and fatal effects of the disease. In such instances the intoxication is cardinally the fact of clinical importance; the infection in itself is a subsidiary element. We have typical examples in diphtheria and tetanus, the diphtheria bacillus and the tetanus bacillus acting in virtue of soluble toxins liberated at the point of invasion, and absorbed into the system. This fact can be experimentally demonstrated in the laboratory, and has received its practical application in serum therapeutics or the antitoxin treatment of disease. We see, therefore, that an infection and an intoxication may go hand in hand on a suitable soil, and that where this occurs the most successful treatment is that directed to protecting the centres of life likely to be affected by the toxins. I have referred to the intoxications resulting from preformed poisons, as, *e.g.*, those present in ordinary food. At the same time there are organisms which appear to require the conditions present in the human body for the elaboration of toxins specifically injurious to the tissues. The seat of this group of organisms is pre-eminently the digestive tract. The organisms may be swallowed with the food or they may be constant inhabitants of the digestive tract, which under certain conditions become abnormally active and form products which, by a direct action on the digestive tract, or by absorption, induce an acute or a chronic form of intoxication. We are familiar with the acute forms of poisoning resulting from, and with the various kinds of enteritis of bacterial

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origin; whilst in infantile diarrhœa we have one of the gravest forms which this combined infection and intoxication may assume.

A large number of intoxications are thus due, directly or indirectly, to the action of living agents; and it may be said in passing that it is more than doubtful whether any infection is unaccompanied by an intoxication to a greater or less degree. The opinion is a growing one that all living agents of disease act mainly in virtue of the toxins they produce, and that all such diseases eventually assume the character of intoxications. There is no more important subject of research at the present moment than that directed to the study of the poisonous products of micro-organisms. The future and most important advances in bacteriology will probably be made in this direction, although many difficulties that do not occur in the case of diphtheria and tetanus have to be overcome. The tendency is to regard an intoxication as the essential phase in any infection, although the experimental proof in many instances is still lacking.

Having referred on broad lines to the relative significance to be attached to the terms infection and intoxication, we may now proceed to the main subject of the present remarks. We are again met by dual terms, which it will be necessary for us to examine in the first place. The terms I refer to are auto-infection and auto-intoxication. Let us first consider these apart, and in this way we will be led to define more accurately the exact scope and limits of our subject. The term auto-infection is not a very exact one, and is used at times promiscuously along with auto-intoxication. It may be used to describe the form and manner of propagation of a disease in the system. Used in this sense the term auto-infection includes the metastatic forms of infective diseases due to the formation of metastatic foci of micro-organisms in various parts of the body. For example, we may instance pyæmia and the multiple abscesses to which it gives rise in various organs. Further, in many infective diseases foci are formed which may be far removed from the primary seat of infection. Thus a pyæmia may occur in the course of enteric fever, and so on. The possibilities for the transference of a morbid agent by the blood and the lymph-stream are numerous, whether it be a tubercle bacillus or a cancer cell. It was customary to use the term auto-infection in another sense, as applied to those cases of infection where it was not possible to obtain a clear understanding as to the mode of development of the infection. We may instance the morbid puerperal processes occurring, as we were once apt to think, without apparent reason. We know now, however, that for many of these conditions the pyæmic cocci are responsible; that they act by gaining admission to the injured surfaces and thence to the blood-stream. In such instances the auto-infection is only an apparent one. On the other hand, we are able to speak with greater definiteness with respect to auto-intoxication, and to define it as a poisoning due to toxins produced by the body itself. Here we are dealing not with intoxications generally (though it is difficult to draw a hard and fast line), but with a group of intoxications, the result of a depraved or abnormal phase in the metabolism of the body. The body is a great chemical laboratory, which is constantly dealing with a variety of chemical compounds, and the processes are of such a complex and unique nature as to baffle largely the most refined methods of organic chemistry employed for their detection. The proteids, carbohydrates, fats, etc., have to undergo many changes in the course of their amalgamation with the tissues of the body. They are ultimately subjected to regressive processes, and are eliminated from the body in the form of relatively simple compounds, such as carbonic acid, urea, and uric acid. This long physiological metamorphosis, with its intermediate products, is at present only known to us in part, and still less can we speak definitely of many pathological phases of metabolism which lead to sundry forms of auto-intoxication. At any rate the chain of events may result in the production not only of useful and indifferent, but also of injurious and toxic bodies; whilst any check to the normal processes of elimination may lead to an accumulation in the system of normal waste products and a consequent intoxication. The most ordinary constituents of the excretions may lead in this manner to an intoxication. CO_2, urea, and bile may respectively lead to asphyxia, uræmia, and colæmia; and in the diabetic coma we have a classical example of an auto-intoxication. Other forms have been added to the list or rendered probable. These instances will suffice to indicate the significance of the intoxications which are due to perverted metabolism in the body. The subject

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is one about which we have still much to learn, and its investigation is beset with much greater difficulties than is the case with regard to the purely parasitic side of disease. The developments which have been made are due to the more exact knowledge now possessed of the chemistry of the blood and the urine. A most useful clue to the organic chemistry of the body lies in the urine, and the study of its constituents in health and disease, and notably as regards the auto-intoxications.

The doctrine of auto-intoxication is a comparatively new pathological conception. Research is becoming more and more concentrated on the study not of the morphology, but of the physiology of the cell, whether animal or vegetable, and the investigation of the chemical processes which have their seat in the cell. This has contributed to the development of the doctrine of intoxication as already indicated. It is no longer a theory; it is an established fact that a poisoning of the organism can occur through the products of its own metabolism. And these metabolic processes have their seat in the several cells of the tissues and organs. Consider for a moment the chemical processes which come into play in the passage of food from the mouth to the large intestine. There are the numerous secretions and ferments which manipulate the food constituents and transform them into assimilable modifications.

The attack begins in the mouth, is continued in the stomach, and centres in the chemical processes of the small intestine. The complex action of the liver and pancreas come here into play, and the digestive action on the proteids, carbohydrates, and fats is completed, the unused remainder passing to the large intestine. In addition we have through the whole course of the intestine the complicating action of numberless bacteria, and notably in the large intestine. Following digestion we have the phenomena of absorption and assimilation, and finally a number of complex chemical processes which have their seat in the various cells of the body—oxidations, reductions, dissolutions, syntheses, and ultimately the chemical modifications preparatory to the elimination of the waste products, resulting in urea, uric acid, salts, etc.

There occur, however, subsequent to assimilation and previous to elimination, a most important chain of events, which constitute the most vital phase of the metabolism of the body. It is their study which forms the most important chapter in physiological chemistry—namely, the study of the intermediate products of metabolism. The products to which I refer are bodies which, under normal conditions, are absent from or only sparingly represented in the excretions, although under pathological conditions, about which we know little, they may be produced and absorbed in excess. We have examples of these in creatin, cystin, oxalic acid, grape-sugar, lactic acid, etc. Under normal conditions these bodies, when produced, undergo further changes, it may be, of oxidation and reduction, or are combined with other substances to form inert compounds. In this way their presence is but temporary, and without inconvenience to the tissues and organs in the exercise of their functions, but circumstances may arise which make their presence felt. It is not my intention to take up your time with chemical details and nomenclature, but it may be interesting to refer for a moment to the proteids and their fate in the body, as the subject of auto-intoxication is intimately bound up with the products of proteid metabolism. We traced the proteids to their transformation in the digestive tract into soluble modifications or peptones. In the intestinal wall they are regenerated into albumen, and subsequently pass into further modification dependent on the action of specific cells. Thus, *e. g.*, the proteids appear as casein in the milk glands, as a constituent of hæmoglobin in the blood, of mucin, keratin, etc. These synthetic processes are all based on the peptones absorbed. They are followed by regressive changes, of which it is mainly the end products that are known to us. (1) Urea; there are probably a number of intermediate stages in its formation. (2) Ammonia. (3) Uric acid. (4) Kreatin and kreatinin as present in muscle and urine. (5) Hippuric acid. (6) Urobilin. (7) Aromatic bodies, the phenol, phenyl, and indol group; a number of these arise in the course of the putrefactive processes occurring in the intestine. (8) Sulphur; almost all is excreted in the form of various compounds by the urine.

The intermediate products of proteid metabolism are, as I have said, imperfectly known, and as to these products are ascribed some of the main features of auto-intoxication, you will see at once that we are on difficult ground. The most

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active propaganda for the doctrine of auto-intoxication has been made in France by Bouchard and others. The French observers have sought to support their theories by experimental data which leave nothing to be desired as regards quantity. Gautier notably has devoted himself to a chemical study of the intermediate products of metabolism, and has differentiated a group of bodies to which he has applied a generic name which will be familiar to you—leucomaines. And as they have been considered to be of pathological significance, it will be proper to devote a few words to their consideration. The leucomaines have been described as alkaloidal products of physiological change. They are basic bodies formed in the living tissues during normal life, and are regarded as due, not to the action of bacteria, but to the activities of the normal tissues. In this respect they are to be distinguished from the ptomaines formed in the course of putrefaction. They are physiological products. Gautier considers that they are formed in the normal tissues continuously in the same way as urea and CO_2, and likewise at the expense of the nitrogenous elements. It is possible that they are antecedents of the end products of metabolism. Bouchard has explained their presence in the urine by assuming that they were originally formed in the digestive tract. The true leucomaines, as described by Gautier, do not, however, fall into this category.

The leucomaines form two broad groups—(1) the uric acid group, and (2) the kreatinin group. A typical example of the former is adenin and also xanthin, and of the latter arginin. The happy hunting grounds for the toxic leucomaines have been the muscles and urine, although Gautier states that normal human saliva contains a number of toxic substances. He regards them as products of the regressive metabolism of proteids. Bouchard considers, therefore, that if they are retained in the body they may give rise to an auto-intoxication. This view has not met with much support outside France as regards the bodies in question, although without in any way affecting the validity of auto-intoxication as a definite factor in the production of disease. There can be little doubt that normal metabolism gives rise to substances which are in themselves toxic, but which find no opportunity to produce such effects. The fact that they are usually innocuous may be attributed to various causes. They are formed in small quantities and they are quickly diluted, or are rapidly transformed into other compounds and undergo a rapid process of elimination. If, however, there be a failure of any one of these factors, an accumulation of these bodies may occur which leads to an auto-intoxication as a result of their passage into the blood, with a consequent toxæmia. The changed quality of the blood converts it into a morbid influence, which makes itself felt in all the vital centres in an acute or chronic fashion.

The possible forms which auto-intoxication may assume have been best classified, I think, by Albu. Albu distinguishes the following possible forms of auto-intoxication:—(1) That resulting from a failure of function in certain organs, as occurs in the case of myxœdema, pancreatic diabetes, acute yellow atrophy of the liver, and Addison's disease. The failure in function has, as a probable result, the non-neutralisation of poisonous metabolic products. (2) That resulting from general metabolic anomalies, whereby the intermediate metabolic products enter the circulation, as occurs in the case of gout and diabetes. (3) That resulting from the retention of metabolic products in various organs, *e.g.* the symptoms after acute burns, CO_2 poisoning, and uræmia. (4) That resulting from the over-production of physiological and pathological products, instances being acetonuria, cystinuria, and diabetic coma. The classification would, however, be incomplete without reference to one of the main seats of auto-intoxication — the digestive tract. Acute and chronic digestive disturbances undoubtedly determine one of the commonest phases of auto-intoxication. We see, therefore, that auto-intoxications are of a protean character. They may start from the skin, as in the case of burns; from the lungs, as in the case of CO_2 poisoning; from the kidney, *e.g.* in uræmia; from the suprarenals, as in Addison's disease; from the intestine, as in various nervous phenomena; whilst the liver, pancreas, and thyroid must equally be held responsible for grave systemic disturbances.

In view of these facts the question is a natural one—why are established cases of auto-intoxication not of more frequent occurrence? I think the answer to that is to be found in the remarkable regulatory and self-defensive forces of the body, whereby noxious agents are neutralised or rapidly eliminated. The skin is one of the most important

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eliminating channels in health and disease, and by its agency normal and abnormal metabolic products are being constantly excreted. The lungs deal with gaseous and volatile impurities both in health and disease. This is particularly marked in diabetic and in septic conditions. The kidneys, however, form the most effective channel of elimination, and free the body from the most dangerous toxic substances. It is also probable that the kidneys act directly and neutralise a number of poisonous bodies. If they fail to do so we have symptoms of a toxic nephritis, ending usually in a necrosis of the kidney substance. This is frequently observed in infectious diseases. The intestine constitutes another important safety valve by the direct elimination of noxious substances, as well as possibly by disposing of urea in the shape of ammonia. And one of the most important protective forces in the body is undoubtedly the liver. One of its most important functions is to neutralise the toxic substances which pass to it from the intestine. Consequently any disturbance in its function inevitably reacts on the whole system. I think that one of the most striking descriptions of the functions of the liver is that which describes it as a filter placed between the intestine and the heart.

From these brief observations it will be seen that the normal defensive forces of the body are considerable as regards an auto-intoxication resulting from its metabolic products. The symptoms of auto-intoxication may express themselves in various ways. (1) Skin: anæmia, colour changes, *e.g.* bronzing, icterus, exanthemata, urticaria. (2) Intestine: symptoms of a more general character, *e.g.* loss of appetite, headache, migraines; and also local, *e.g.* flatulence, colic, and diarrhœa, etc. (3) Kidneys: albuminuria, hæmaturia, hæmoglobinuria, oxaluria, etc. (4) Nervous system: this develops a most important chain of symptoms—headache, giddiness, faintness, insomnia, coma, melancholia, and hypochondriacal conditions; these affecting ultimately the heart and breathing. (5) Blood anomalies, *e. g.* chlorosis, anæmia, diabetes, uric acid diathesis and its consequences.

The intestinal tract is one of the main channels of intoxication. We have occurring in the digestive tract—(1) the processes of digestion; (2) fermentative processes due to the action of bacteria and of a twofold character—of the nature of fermentation and of the nature of putrefaction, *i. e.* carbohydrate and proteid decompositions. Both are constantly occurring. Bacteria were formerly supposed to be essential to the digestive processes, but this has been proved to be unlikely. The decompositions they give rise to result in products which cannot be regarded as of any special nutritive value. The ideal digestion would therefore be an odourless one. The seat of carbohydrate decomposition due to the action of bacteria lies mainly in the stomach and small intestine. The seat of proteid or putrefactive decomposition lies mainly in the large intestine.

The carbohydrate fermentations, *e.g.* lactic acid, butyric acid, acetic acid, and yeast fermentations, are all due to microbes, and result in the production of formic, butyric, lactic, succinic acids, etc. Curiously enough, the bacteria in the small intestine appear also to produce a certain quantity of alcohol. Further, we have various decompositions which yield gases such as H, CO_2, and at times CH_4. These are neutralised, absorbed, and oxidised. The gaseous development in the stomach due to fermentations may be considerable, as in dilatation of the stomach. In the course of proteid decomposition we have numerous products formed, such as (1) NH_3, N, CO_2, H_2S, mercaptans; (2) leucin, amido-acids; (3) phenyl, indol, skatol, etc., or aromatic bodies. Amido-acids are normally absorbed in the intestine. The aromatic bodies pass into the blood, and are eventually discharged as innocuous combinations from the system. They are always increased in putrefactive processes, and form an indication of the extent of putrid decomposition occurring in the intestine. A further product, acetone, is found in the intestine. All the above products are usually eliminated without producing abnormal reactions. If they accumulate we have gastric and systemic disturbances—an auto-intoxication. Basic bodies (ptomaines) and bacterial toxins may arise. The most poisonous are the bacterial toxalbumens. These, rather than the ptomaines, are responsible for the acute symptoms of food-poisoning. If we turn to the liver, we find that failure of the bile favours putrefactive processes occurring in the intestine, whilst the presence of bile in the blood causes toxic symptoms. The liver affords protection against the poisonous products of intermediate metabolism. One of its most important functions is the neutralisation or disposal of various poisons, organic or inorganic.

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A disturbance in its function, therefore, opens a door to intoxications of various kinds. I may add that the glycogen appears to play an important part in these functions of the liver. The liver is one of the most active organs in the body, both in health and disease, and its insufficiency undoubtedly favours auto-intoxication from the intestine. As a result of chronic constipation, various decomposition products may be formed which act deleteriously on the system, but these need not detain us.

The individual susceptibility to such poisonous products varies. The greatest susceptibility is exhibited by the nervous system of children, which is peculiarly sensitive to the action of toxins, and this presents itself in the form of cramps and convulsions. There is probably a close connection between these symptoms and disturbances occurring in the digestive tract. Intoxications in infants may readily be produced, followed by gastro-enteritis and death. This may supervene from artificial or improper foods. The fermentations which may occur in the stomach can likewise lead to many symptoms of auto-intoxication, and vegetable foods favour more particularly the gaseous fermentations.

Most forms of auto-intoxication are accompanied by cerebral and nervous symptoms, and these may be of an acute or a chronic character, and lead to psychical disturbances and certain forms of melancholia. Skin affections may arise, *e. g.* itching in uræmia, boils, gangrenes, etc.; urticarias following consumption of certain foods, and so on. I am afraid that in combating such conditions too much reliance is not to be placed on the use of intestinal antiseptics. I consider it almost hopeless to produce any inhibitory effect of value on the bacterial decompositions occurring in the lower part of the intestine, or to attempt with any hope of success a disinfection of the digestive tract. I am aware that a number of substances have been recommended for this purpose, but their efficacy is doubtful. Much better results are at present to be obtained by attention to diet, adequate purgation, and other means to remove the causes at work. We have yet to find the means of adequately treating the acute forms of auto-intoxication resulting from food and other poisons.

This brief survey would be incomplete without reference to certain other disorders in which auto-intoxication is supposed to play a prominent part. We have, *e. g.*, the diabetic coma in which β-oxybutyric acid appears to be one of the determining factors; whilst in cancer there is evidence of a chronic auto-intoxication deleterious to the whole cell-life of the body. In such cases we have possibly to deal with rapid destruction of cell-tissue elements along with an accumulation in the system of the resulting metabolic products. If we turn to the urinary secretion we have forms of hæmoglobinuria due to the action of toxins which produce a rapid dissolution of the blood-corpuscles. Much attention has recently been devoted to the study of these blood poisons or hæmolysins. There can be little doubt that many of these have their source in the action of bacteria, not only in the system, but possibly also in the digestive tract.

Our knowledge of the poisons present in the urine still leaves much to be desired. What is at present known is not sufficient to elucidate the various phases of auto-intoxication. The attempt has been made to bring chlorosis within the sphere of auto-intoxication, and some of the facts favour such a theory. As regards pernicious anæmia, the symptoms have been referred to increased hæmolysis. It has been thought that as the result of gastric disturbances poisonous bodies are formed which, on absorption, attack the blood-cells. Dr. W. Hunter considers this form of anæmia to be a special infective disease associated frequently with infection of the alimentary tract, and at times with oral sepsis.

An interesting observation is with regard to fatigue fever. For example, soldiers on the march develop what appears to be a form of auto-intoxication. It may possibly be a decomposition of the blood by absorption from the overtaxed tissues of poisonous substances and waste products.

I think I have now said enough. The facts which we have considered largely justify our ranking auto-intoxication as a definite pathological factor, and in attributing to it an important *rôle* in the production of disease. The condition is frequently a grave one on account of the profound effects almost invariably produced on the nervous system. Chemical and biological research promise to throw still further light on this important element in disease, and it is to be hoped that the study of auto-intoxication will be actively pursued both from a clinical and an experimental standpoint.

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THREE LECTURES ON ACUTE APPENDICITIS.

LECTURE III.

GENERAL DIFFUSE PERITONITIS DUE TO APPENDICITIS.

Delivered at the Polyclinic on March 6th, 1903.

By HAROLD L. BARNARD, M.S., F.R.C.S.,
Assistant Surgeon to the London Hospital.

THE subject of general peritonitis in appendicitis is of the greatest possible interest and importance, for practically all the cases which die from appendicitis do so because general peritonitis has supervened. If we wish, then, to diminish our mortality we must do so by studying the manner in which general peritonitis is produced, in order that we may if possible prevent its origin. When once general peritonitis is established the prognosis is exceedingly grave, but I shall hope to tell you of a method which gives some prospect of success even in this serious condition of affairs.

Nomenclature.—The term general peritonitis has been used in a very vague and indefinite sense, so that whilst so skilled an operator as Sonnenburg, of Berlin, reports that seventeen consecutive cases of this malady died under his treatment, other people claim almost any percentage of successes from 25 up to 75 per cent. It is, of course, clear that these surgeons are using the term "general peritonitis" in very different senses. We must therefore begin by defining what we shall take general peritonitis to mean. In the first place, by general it is implied that every part of the peritoneum is inflamed, and whilst this is a condition which can only be certainly confirmed at an autopsy, yet we may diagnose it if every part of the peritoneum which is accessible to our touch is tender and painful. By the term "diffuse" we imply that the peritonitis is not localised, as it is in the case of appendix abscesses. There is no line of demarcation between the area of peritonitis around the appendix and the rest of the abdominal cavity, as there is in the latter condition. In the condition of general peritonitis no lymph barrier has been formed, and it is this barrier which circumscribes the inflammation in the more favourable termination of appendicitis. Many people hold that general peritonitis must be suppurative, but this is not at all necessary, for, as I shall point out to you later, the worst forms of peritonitis are those in which no pus is formed.

We will now consider the *signs and symptoms* on which a diagnosis of *general diffuse peritonitis* can be made. The entire abdominal wall is tender on palpation. This tenderness extends not only over the entire anterior surface of the abdomen, but also back into the loin at the sides as far as the point at which the peritoneum is reflected on to the colon. The abdominal wall is as a rule rigid, and there is no movement on respiration. It presents, moreover, a smooth aspect, with well-marked veins, which is very characteristic of general peritonitis. Distension is present in the worst cases, and when it is due to paralysis of the gut by the inflammatory process it is, as you will easily appreciate, a most serious sign. But in many cases the distension is due to obstruction of the intestine by the matting of the coils of gut around the appendix, and when this is the case it is not of such serious import. The late Mr. Greig Smith drew attention to a most valuable sign of general peritonitis. He recommended that the abdominal wall should be explored methodically with a stethoscope for five minutes, and if during the whole of this time no gurgle was heard in the abdominal cavity indicating a peristaltic movement of the gut, then it might be safely concluded that the whole of the intestine was paralysed. This sign I have found of the greatest value in many doubtful cases. The face in general peritonitis is of a greyish colour and sunken. It presents a great contrast to the flushed face of pneumonic diseases. In general peritonitis the eyes are sunken and surrounded by rings. The tongue is, as a rule, small, shrivelled, and dry. The teeth are covered with sordes. We may largely attribute the condition of the tongue and mouth to the fact that the secretion of saliva is practically suppressed in consequence of the great deficiency of fluid in the body, which at the same time makes itself evident in the shrivelled tongue and the sunken eyes. Vomiting is practically always present; it is profuse and persistent, and towards the end it may become dark-coloured and even fæculent in odour. The pulse is, as a rule, as rapid as 130 to 160 per minute. In some few cases of general peritonitis the pulse-rate is below 100 per minute, and yet at the operation the entire peritoneal cavity

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is found full of pus. I believe the explanation of this surprising condition of things is that the leucocytes which form the pus have so effectually stopped up the stomata of the peritoneum that practically no absorption of toxins is going on. I shall, however, refer to this process in more detail when dealing with the pathology of peritonitis. The temperature in general peritonitis is little if at all raised. Indeed, the severity of the condition may be rather taken to be inversely as the height of the temperature, so that in really serious cases the temperature is subnormal. It is only at the onset of the attack, and whilst the process is still localised, that we ever see temperatures of 103° to 104°. The patient is practically always constipated, and will react only with difficulty to purgatives or enemata. In the treatment of general peritonitis, to which I shall draw your attention later, purgatives form an important part, and I may say now that we have come to estimate the severity of a case by the quantity of calomel required to open the bowels. If the bowels are not constipated, or are easily made to work with an enema, the case is one of good prognosis.

The Causation and Pathology of General Peritonitis.

General peritonitis is due to the invasion of the peritoneum by an army of micro-organisms advancing from the inflamed appendix. The peritoneum is provided with certain measures of defence with which it opposes, and in many cases arrests, the advance of these bacteria. We shall, then, consider in detail (1) these means of defence, and shall proceed to consider in the next place (2) the causes which lead to their failure to localise the infection and convert the case into one of an appendicular abscess. Lastly, we must consider (3) the state of affairs which results from this failure to arrest the advancing infection, in order that even when general peritonitis is present we may combat these results with (4) a rational treatment.

1. *The Measures of Defence of the Peritoneum.*—When a perforation suddenly occurs in an appendix and a few minims of pus are thrown into the peritoneal cavity, the micro-organisms in that pus would within a very few hours spread to the furthest recesses of the peritoneum were they not opposed by the measures to which we are about to refer. Practically immediately the neighbouring peritoneum pours out a copious flood of *antibacterial serum.* A similar effusion of serum is seen when infection in the end of the tibia or femur is threatening to invade the knee-joint, or in the pleural cavity when a subphrenic abscess is threatening to perforate the diaphragm. In the course of some hours *the leucocytes* begin to be poured out in vast numbers from the subperitoneal vessels and to permeate the pools of serum which are resisting the advance of the micro-organisms. These leucocytes appear to act in several ways in arresting and limiting the infection. They are probably phagocytic in action, and actually devour some of the micro-organisms. But in another way they act even more powerfully in protecting the system from the toxins of the micro-organisms by stopping up one by one the stomata in the peritoneum. A healthy peritoneum is so perforated by stomata which lead from its cavity into the neighbouring lymphatic and blood-vessels that it may be compared very aptly to a sieve, and it is capable of the most rapid absorption. If this absorption were to continue after the invasion by the micro-organisms, in a few hours the patient would die poisoned by a massive dose of toxins; and, indeed, this does occur in rare instances. But first delayed by the antibacterial serum and then attacked by the leucocytes, the formation of toxins is retarded, and their absorption is finally prevented by the blocking up of all the neighbouring stomata by the leucocytes, so that the wall of the cavity in which the pus is contained becomes practically impermeable. The last means of defence of the peritoneum is the formation of *the lymph barrier;* behind the two lines of defence to which we have already referred the intestines are matted together by masses of living lymph, which consolidates them into a barrier well adapted to resist the advance of the infection. For the formation of this barrier two or three days are necessary, but after that time it is rapidly consolidated into definite adhesions, and in the course of a week or so the lymph is actually vascularised, and a layer of living tissue circumscribing the infection is absolutely complete. On the side of the barrier towards the appendix the peritoneum has been given over to the micro-organism, and the serum and leucocytes in that area are now converted into a pond of pus swarming with bacteria. But all egress from this circumscribed space is practically blocked, and we have indeed a localised abscess. The general peritoneal

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cavity behind the barrier is now safe from infection, and if serum has been poured out there it is now re-absorbed without passing on to the formation of pus.

In cases of general peritonitis all these means of defence have broken down, and the infection has advanced across the peritoneal cavity. We accordingly find *different varieties of general peritonitis according to the degree of the failure in the defence.* In some cases the lymph barrier has actually been formed, and the process localised, when some indiscreet exercise or some unwise purgative has broken down the lymph barrier and allowed the virulent pus to flow through into the open peritoneum beyond. If the serum and the leucocytes are poured out but no lymph barrier is formed, then suppurative peritonitis spreads all over the peritoneum. The formation of pus is comparatively a favourable sign in diffuse peritonitis, and cases in which it is formed are not so fatal as those in which the serum is poured out, but there is no diapedesis of leucocytes, either because the process is too rapid or because the patient fails to react. In these cases we find blood-stained serum, which is only a little turbid; not a trace of lymph is seen upon the intestines, and the surface of these is roughened and eroded. So that not only are the stomata not occluded, as they are in a suppurative case, but the very endothelial covering of the peritoneum is removed, so that the most rapid absorption of the toxins takes place from the peritoneum. In such cases of peritonitis we therefore find the signs of profound toxæmia; the pulse is usually as rapid as 170 and extremely feeble; the patient is cold and collapsed, and death supervenes within a few hours. A few cases have been recorded where even the effusion of antibacterial serum broke down, and where a dry peritonitis, running its course in twenty-four to thirty-six hours, carried the patient off with signs of the most profound toxæmia. (2) *The reasons why the serum, the leucocytes, and the lymph barrier fail to arrest and localise the peritonitis are as follows:*

In the first place the dose of pus which suddenly enters the peritoneal cavity is probably the most important cause. Where the micro-organism has to grow through the wall of the appendix the invasion is of necessity slow, and there is ample time for measures of defence, but where an appendix distended with pus suddenly perforates, and a drachm or so of virulent pus enters the peritoneal cavity, there is great risk that it may not be localised. In general peritonitis cases, accordingly, we nearly always find a perforated appendix.

The character of the micro-organism is also a most potent factor. Some micro-organisms are of such extreme virulence that they can perforate the wall of a living appendix, and can then spread far and wide through the peritoneum, overcoming all resistance. Some twelve months ago I operated on such a case of appendicitis, where the appendix did not contain a calculus, nor was there any stricture; but the whole structure, though still alive, was acutely inflamed, and the process had extended throughout the whole peritoneal cavity. In cases such as this we must regard the cause of the general peritonitis to be the virulence of the micro-organism. In other cases the continuous spread of the micro-organism appears to be due to its power to dissolve the lymph laid down as a defensive measure by the peritoneum. Thus in another case I had under my care some time ago, on which I operated forty-eight hours after the onset of the attack, I found the peritonitis fairly restricted to the right iliac fossa, but there was no formation of lymph. The appendix was removed, and the area of peritonitis packed off with gauze; but although the man lived eight days the peritonitis did not cease to spread steadily during the whole of that time, and at the post-mortem we found a total absence of lymyh in the peritoneal cavity, and even in that part of it which I had packed off with gauze.

The position of the appendix in relation to the mesentery of the small intestine appears to me to be an important cause of general peritonitis. When the appendix lies behind the mesentery and the small intestine, any abscess it may form is readily localised against the posterior abdominal wall. But when it lies in front of the mesentery and the small intestine, with its end in the central region of the abdomen, then should a perforation occur it is far from the abdominal wall, and it at once infects the whole of the central region of the abdomen, which seems to be the most dangerous and to produce the greatest degree of shock. In only two cases have I definitely localised the appendix in front of the mesentery and the small intestine, and in both these instances general peritonitis resulted when the appendix became inflamed.

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In some cases the fault appears to lie with the patient. There are some people who appear to react very inefficiently to bacterial invasion, and these people are often some of the strongest and most athletic members of society. One has often noticed in hospital life how the big athlete appears to be susceptible to every form of bacterial invasion. He suffers from hospital sore throats, from whitlows, and catches all the infectious diseases which he encounters in the hospital; whilst some anæmic and apparently feeble student will go through his entire course at the hospital without a single day off duty for ill-health. In the same way patients exhibit very different powers of reaction to peritoneal infections. Closely connected with this subject is the interesting question of the immunity which the peritoneum acquires after one attack has been safely passed. It is the absence of this immunity which makes a first attack so much more fatal than any subsequent one, and this fatality, as we have seen, is due to the greater frequency of general peritonitis in first attacks. The immunity of the peritoneum after the first attack may be partly explained by the formation of adhesions which mechanically localise the process from the very start; but there is no doubt that the immunity is in part at least actual, and that the peritoneum is more or less indifferent to the presence of the micro-organism. It is possible that in these cases the serum which is poured out is of higher antibacterial and antitoxic action. A common cause of the failure of the lymph barrier to circumscribe the peritonitis is the occurrence of trauma, and this is of great practical importance, as it shows the necessity of keeping all cases of appendicitis absolutely quiet. Everybody must have known of cases of appendicitis which progressed favourably for two or three days, in which the patient then got out of bed, relapsed, and died of peritonitis shortly after. In these cases we can only suppose that the movements broke down the lymph barrier and allowed a flood of pus to pour through the rent into the general peritoneal cavity. In many cases a similar result has followed the administration of a violent purge, and here no doubt the violent peristaltic contraction of the intestines separated one coil from another, and in this way broke the continuity of the lymph barrier. Purgatives, then, should be absolutely avoided in all localised cases of appendicitis, although, as you will see, they form a most important part of the treatment when the peritonitis has spread to all parts of the cavity.

We must now consider (3) *the condition of things which is brought about by the failure of the peritoneum to arrest the spread of infection.* In the first place the serum, which has been converted into pus by its invasion by leucocytes, stands about in ponds in the peritoneal pouches to which I drew your attention in my last lecture. This pus is of a most virulent character, and is charged with the toxins manufactured by the micro-organism. So virulent is it, indeed, that when in releasing it we allow it to flow over the raw margins of our wound it kills the tissues so that they are reduced to a sloughing condition. It is an astonishing fact that a patient may hold within his abdominal cavity some pints of such a virulent fluid, and that yet his condition may not be profoundly serious. The explanation is, as I have said, that the stomata of the peritoneum in such cases are all effectually blocked by the leucocytes, and, as we shall see later on under the head of treatment, in emptying these collections of pus we must disturb these natural defences as little as possible. In spite of these means to prevent the virulent poisons manufactured by the micro-organism from reaching the general circulation, there is nevertheless in general peritonitis a very serious degree of toxæmia. This toxæmia shows itself in the ashy colour, the condition of collapse, the feeble heart, and the extreme nervous exhaustion. Another very serious result of general peritonitis is the paralysis of the coils of gut which are exposed to its action. This fortunately does not always occur, for it will be readily understood that when the inflammation is sufficiently severe to penetrate the wall of the intestine and paralyse it, there is little chance that the patient will recover. The paralysis of the intestine leads to a stagnation of its contents, which decompose and distend the intestine by a copious formation of gas which cannot be got rid of by the paralysed gut. This intestinal decomposition adds to the general toxæmia. Finally—and perhaps this is the most immediate danger of general peritonitis—there is a tremendous shortage of fluid in the body. It must be remembered that for three or four days not only has the patient not taken any fluid by the mouth, but during that time he has expended his stock of fluid in supplying the excretion of urine, sweat, and the copious vomiting. It

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is well known that patients suffering from general peritonitis will lose a stone or more in weight in a few days, and this loss of weight is chiefly due to deficiency of water. We see this fact plainly indicated in the shrivelled tongue, the depressed eyes, and the drying up of the secretion of saliva in the mouth. The deficiency of fluid in the body must also have a most profound effect upon the circulation, just as it has in a similar condition in cholera.

(4) *Treatment. The Rational Treatment to combat the above results.*—First we must drain the peritoneal pouches, which are filled with ponds of pus. After having tried practically every means of doing this except evisceration, I have come to the conclusion that the rational thing to do is to empty these abscess cavities with the greatest gentleness and with the least disturbance of the natural defences which shut their contents off from the vascular system. We went very thoroughly into the anatomy of these peritoneal pouches in our last lecture, and I then indicated that by three incisions they might all be emptied with the least possible disturbance of their walls. I must refer you for these incisions to the previous lecture, but I may say that they are a median one above the pubes through which the pelvic pouch and the iliac fossæ can readily be reached, and two lateral ones in either loin which give access to the subphrenic spaces on each side, and on the right also allows the subhepatic space to be efficiently drained. The appendix, if it is encountered, should be removed. I am strongly opposed to irrigating the general peritoneal cavity in these cases, for in the first place the toxin which was concentrated in the peritoneal cavity is well diluted and flushed into the general system. Then I believe the mechanical washing opens up the stomata which have been closed by the formation of lymph by the leucocytes. It often happens, moreover, that the fluid employed is not of the correct temperature, and coils of gut acutely inflamed are most sensitive to lavage with water that is too hot or too cold. As a result of some considerable personal experience of irrigation, I may say I gave it up because so many patients collapsed entirely after its employment.

Secondly, the use of boric acid. I should like to begin by saying that I have only employed this measure in two cases, but that in both these cases it was successful, and at any rate it does no harm. It seemed to me that in those cases of peritonitis in which the healthy peritoneum was not shut off from the micro-organism by lymph in a similar way the micro-organism was exposed to attacks by the surgeon, could he discover an antiseptic which would not also prove fatal to the patient. Boric acid naturally presented itself as fulfilling these conditions. Even in water it does not form more than a 5 per cent. saturated solution, and it is certainly far less irritating to living tissues than the virulent toxins produced by the micro-organisms upon whom it acts. I accordingly had long sausage-shaped bags made of gauze, the end of which for a couple of inches or so was packed with boric acid powder; the whole was sterilised by heat. In two cases, one of which was diffuse peritonitis without adhesions, I planted these bags of boric acid in the various pouches of the peritoneum, the tail of the bag acting as a gauze drain. After three or four days, when the bag is pulled out it is found to be empty, and I believe that in both these cases, although suppuration was not stopped, the abscess cavity more rapidly cleaned up, and they healed more quickly. A more extended experience is, however, necessary before speaking definitely upon this subject. At any rate, as I have said, it does no harm, and it is well known that even small quantities of boric acid in solution will inhibit the growth of micro-organisms even when it is not strong enough to kill them.

The third means of treatment we are employing at the London Hospital for general peritonitis cases is to administer a grain of calomel every hour until the bowels are opened, and then to maintain a moderate diarrhœa. In some cases as much as 40 and 50 grains of calomel have been administered, and in one case 72 grains were given. Mr. Lawson Tait years ago advocated the use of saline purges in general peritonitis, but he gave the preference to magnesium sulphate. I prefer calomel, not only because it is easily administered and the patient cannot vomit it, but also because I believe that given in small and repeated doses it is the most powerful antiseptic of the entire alimentary canal that we possess. For instance, it is well known that calomel administered in small doses repeatedly to children produces grass-green motions. This result appears to be due to the action that calomel has exerted on the butyric acid fermentation in the alimentary canal. The biliverdin of

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the bile therefore passes from the liver to the rectum without being reduced to bilirubin, as is usually the case. The reason for administering purges is to prevent the dilatation and paralysis of the gut, with the consequent decomposition of its contents, to which I have already referred.

Finally, we employ massive saline subcutaneous transfusions, and it is the introduction of this line of treatment at the London Hospital which has entirely changed our point of view with regard to cases of general suppurative peritonitis. Previously we regarded these cases as practically hopeless, and approached them with a feeling of helplessness which was very depressing. It was only because now and then a case recovered in spite of all expectations that we continued to offer the parents this forlorn hope. I may say that we now approach cases of general peritonitis with a considerable feeling of hopefulness, and with a sense that a good fight will probably save our patients. I must tell you first how this treatment was brought to my notice. The chief assistant to Professor Kocher, of Berne, was visiting the London Hospital, and I was asked to show him round the wards. As we passed from one part of the hospital to another, we compared notes on the surgical matters that had interested us lately, and I asked him if in Berne they knew any cure for general suppurative peritonitis. He was enthusiastic about massive saline transfusion, which he told me he had adopted successfully in two cases. He described to me his apparatus, and told me that he had transfused twenty or thirty pints by four needles placed in the subcutaneous tissues. The apparatus employed by him is a somewhat complicated one, but I show you here to-day a simple and practical form, which is also inexpensive, and which can be obtained and used successfully by anyone. About four feet of rubber tubing, weighted at the end, carries the saline from an ordinary ewer to a glass T-tube, and this is connected by two further lengths of rubber tubing with two stout brandy-syringe needles which can be readily passed below the skin of the thighs. The saline should be of the strength of a teaspoonful to the pint (0·6 per cent.); the water should have been boiled, and in the ewer should remain at a temperature of about 115° F. The ewer should be scalded out before use, and the whole of the tubing should be boiled. To establish a syphon action it is only necessary to put the whole of the tubing into the water in the ewer, and then having clipped the end to draw out the T-tube and needles over the end. The ewer should be placed at the height of one foot above the thighs of the patient. If a greater height than this is employed the distension gives rise to considerable pain. With this elevation two pints of saline will flow into the subcutaneous tissues in an hour through the two needles. It is of course well to clean up the thighs and to insert the needles with all antiseptic precautions, but as long as ordinary care is employed it is not well to waste time in such serious cases in elaborate precautions. I must admit that one or two cases have developed abscesses at the point of puncture, but these cases were of such a serious character that they may consider themselves fortunate to have lived long enough to develop these abscesses, and the latter were after all of a very mild subacute character, and healed up without the least trouble. The quantity of saline infused in this fashion must vary considerably with the size of the patient. A full-grown man can safely take fifteen pints in the first twenty-four hours after the operation. In two little boys of seven or eight whom I recently treated in this way, we infused four pints directly after the operation, and four pints a few hours later. Eight pints is a moderate quantity. The transfusion is repeated day by day as occasion demands, unless the vomiting ceases and the patient can drink freely, or the rectum will absorb a pint six times a day. The effect of this transfusion is really very wonderful; the whole face of the patient fills out, the rings round the eyes diminish or may even disappear, the tongue becomes larger and moist as the secretion of saliva is once more established, the pulse is greatly improved and becomes slower and stronger. In some cases I have seen infusions increase the flow of pus from the abdominal tubes. This saline method is based on many sound reasons. In the first place, as has been pointed out, the body is very short of fluid, and it is a well-known fact in biology that all protoplasm, in order to perform its functions well, must be distended with water. I show you here two flowers, one of which I removed from water some hours ago. You will see that it is drooping and faded, but if I replace it in water, within an hour or two it will look as fresh as its companion. In the same way, when a human being is deprived of water he fades and droops, and, as I

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have pointed out to you, our peritonitis cases are not only taking no fluid for three or four days, but they are constantly losing water from their system through many channels. They cannot drink to replace this water, and saline infusions into the rectum do not always act well, more especially as we wish to keep up a persistent diarrhœa by means of calomel. We can then replace the water that they have lost, and maintain a continued supply of it by these two needles placed beneath the skin of their thighs. In the second place these massive transfusions alter the relation between the vascular system and the pus in the peritoneum. Whilst the vascular system is depleted, there is every inducement for fluid to pass from the ponds of pus in the peritoneum into the vessels around. But when by means of ten or fifteen pints of saline we have thoroughly filled up the vascular system, not only is there less inducement for the toxins to be absorbed into the blood, but a flow tends to be established in the opposite direction; and, as I have told you, some cases in which abscesses in the peritoneal cavity were being drained by tubes have had an increased flow from those tubes when saline transfusion was established.

In the third place the transfusion dilutes the toxins circulating in the blood, and probably in this way diminishes the virulence of their action on nervous tissues, on heart muscle, and on the other tissues of the body. In the last place this flood of saline passing through the body carries out with it, especially through the kidneys, these toxins in a very dilute form, and with the least possible damage to the renal epithelium.

In speaking of the results of this system of treatment, it is very difficult to speak definitely, for the difficulty which we encountered at the beginning of this lecture in defining what should be called general peritonitis makes the collection of a series of cases which would stand that test extremely difficult. And when one is enthusiastic about any form of treatment, there is a very natural tendency to sweep into the results less severe cases. I will, therefore, simply state that in five successive cases of peritonitis under my care in which the inflammation was practically general, only one died; and in this case death occurred one hour after the operation, and before the method of treatment could be got into running order. A colleague of mine who is employing the same line of treatment has had only one death in six cases of general peritonitis. One of his cases, I think, is deserving of especial note. The boy was admitted to the hospital not only distended, but with fæcal vomiting. He required seventy-two grains of calomel in hourly doses to open his bowels and to keep them open. He was transfused twenty-six pints in all on, I believe, four occasions of about seven pints each.

I can definitely state that this method of simple drainage, combined with calomel and massive transfusions, has in my experience proved the most hopeful measure I have yet encountered in the treatment of general diffuse peritonitis. This method has, moreover, made it possible to prolong cases of peritonitis to a point at which two recovered by an actual crisis. This phenomenon, I believe, has not previously been observed, chiefly because hitherto patients have died of toxæmia and deprivation of fluid, and were not able to fight to a finish the actual battle with the micro-organism. In one of my cases, a little boy of eight years of age, we removed the appendix and drained the pelvic abscess, but found the peritoneum generally infected. By means of the treatment I have indicated we kept him alive for ten days, during the whole of which time he was vomiting, his temperature was slightly raised, and the edges of the wounds remained dry and glazed. At the end of these ten days, one afternoon he had a most curious attack of delirium. His pulse fell from 132 to 72 per minute in twenty-four hours; the vomiting ceased, he began asking for solid food, and the wounds in the abdomen took on a healthy appearance.

In a similar case of mine, in a girl æt. 17, the pulse fell from 124 to 96 in twenty-four hours, her temperature came down, and she rapidly convalesced. The occurrence of these crises impresses on us the lesson that ultimately the battle will be fought out between the man and the micro-organisms in the inaccessible recesses of the peritoneal cavity, and that in some cases the micro-organism may gain the upper hand. No treatment will therefore always be successful in general diffuse peritonitis, but whilst the battle is still undecided, and whilst the result is still in the balance, there is no reason why our patient should die of depletion of fluids or of paralytic dilatation of his intestine.

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WITH MR. D'ARCY POWER

IN THE OUT-PATIENT ROOM AT ST. BARTHOLOMEW'S HOSPITAL.

June 15th, 1903.

(*Concluded from page* 288.)

2. *Successful Wiring of an Ununited Fracture of the Humerus.*—The next patient had an ununited fracture of the humerus, and Mr. D'Arcy Power said,—I think many of you will recollect this girl of fourteen who came here in January with an ununited fracture of her right humerus. She told us that it was broken eleven years since, and that in spite of nine operations by competent surgeons it had never united. I rather dissuaded her from having anything more done, as her arm was well developed and useful, for she could raise it by a swinging movement. Her friends, however, were anxious that another attempt should be made to secure union, and I took her into the hospital on February 4th, 1903. When the fracture was exposed in the operating theatre it was found that the two ends of the humerus had formed a perfect false joint at the junction of the middle with the lower third of the bone. There was a capsule containing fluid, and the ends of the bone formed a ball-and-socket joint, being covered with fibrous tissue.

The ends of the bone were removed with considerable difficulty, as the bone was densely hard, and, after drilling, they were brought into contact with two of the stout silver wires we use for joining fractured patellæ. The arm was put up on a splint for a month; it was then dressed, and the sutures were removed from the skin wound, which had healed perfectly. This skiagram, taken on the 15th of March, shows that there was some attempt at repair, as the bridge of bone connects the two fragments; and the second skiagram, taken in June, shows that this repair has continued, for the connecting bridge of bone is much more marked. I have sent for her to ascertain her condition at the present time, as I have not seen her since she went out of the hospital. You will notice she has got a perfectly movable elbow-joint. She has atrophied muscles, but that is only natural from having had the splint on since February, and they will recover themselves in a month or two. There is no movement between the two ends of the bone, so I think it will remain secure.

3. *Recovery after Two Operations for Intussusception.*—The next patient is a baby who recovered after two operations for intussusception, a sufficiently formidable ordeal. This condition, as you all well know, is very much more fatal than it would be if mothers were taught to apply early for relief, and if students understood that an intussusception may be present without any tumour being felt through the abdominal walls, and also that every tumour within a baby's abdominal cavity is not an intussusception. The child was in hospital and in Lawrence Ward under the care of Mr. Walsham from January 16th to February 16th. Mr. Walsham performed an abdominal section upon him for the relief of acute ileo-cæcal intussusception. He was seven months old when he was readmitted to Lawrence Ward under my care on April 23rd, 1901. Two days before he had been suddenly seized with "cramp" in the stomach, which made him cry continuously. He vomited every time he was suckled, and he had passed a little blood and slime by the anus. The same symptoms continued through the day following the onset of the attack, except that the child appeared drowsy and passed neither blood nor mucus by the bowel. On April 24th he still continued to vomit, and passed blood mixed with mucus on two occasions. When I saw the baby on April 25th he was rather collapsed and very apathetic. The abdomen was distended, and a distinct tumour could be felt in the region of the hepatic flexure of the colon. There was a marked loss of resistance in the right iliac region. A discharge of blood-stained mucus followed the introduction of the finger into the rectum. The scar of a soundly healed incision occupied the middle line of the abdomen below the umbilicus. I made an incision immediately to the right of the scar, and on passing my fingers into the abdominal cavity I felt a distinct tumour just beneath the liver. This tumour was the enlarged and thickened ileo-colic angle, which had been drawn well up above the level of the umbilicus. It was impossible to bring the tumour into the wound, and whilst feeling along the colon to ascertain the nature of the resistance an invagination of the colon into the colon was discovered about the middle of the transverse colon. This intussusception was easily reduced by pressure

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applied distally, and without bringing the bowel out of the abdominal cavity. The ileo-colic angle could be drawn out of the wound as soon as the colo-colic invagination was reduced. The cæcum was then found to be thickened by chronic inflammation, whilst the terminal portion of the ileum lay parallel with the ascending colon, to which it was adherent for some distance. The vermiform appendix was unusually long, but it was quite healthy. There was no ileo-cæcal or ileo-colic intussusception. The adhesions between the ileum and the ascending colon were gently broken down, and it was noticed that portions of the small intestine were adherent to the peritoneal surface in the neighbourhood of the former wound. The entire thickness of the abdominal wound was brought together with sutures of silkworm gut, and the operation was completed in twenty-two minutes. The child never vomited after the operation. Food was given by the mouth four hours after he recovered from the anæsthetic, and the bowels were open naturally on the following day. He made an uneventful recovery, and was discharged from the hospital cured on May 5th 1901. His mother now says he cannot cough very comfortably, and that is probably because the effort to cough drags on the adhesions, which we noticed during the second operation. I would give you a word of warning in these cases, namely, not to take out the sutures too quickly. We had a case eighteen months ago in which we left the sutures for ten days, and then took them out. As soon as the sutures were removed the wound gaped, and the small intestines bulged out of the wound. They had to be put back under anæsthesia and the wound was again sewn up. Therefore leave the sutures in as long as you can—at least a fortnight or three weeks after the operation.

4. *A Cancer of the Breast treated by Double Oöphorectomy.*—The last patient I asked to come up to-day to see us is a case of cancer of the breast relieved by double oöphorectomy. You have seen her several times already, as she has presented herself with great and commendable regularity. She is a widow, æt. 43, who has been a hospital nurse. She was admitted under my care into St. Bartholomew's Hospital on April 21st, 1902, suffering from a carcinoma of the right breast, which she had noticed for a year. At the time of her admission a large mass of carcinoma involved the breast and there were secondary growths in the skin, in the axillary glands, and in the infra-clavicular fossa. There was also a suspicious swelling in the left breast. The right breast was immovably fixed to the deeper tissues. You will remember that when I showed her at consultations my colleagues agreed unanimously that the case was inoperable, but they fell in with my suggestion that I might perhaps give her relief by removing the ovaries. I performed double oöphorectomy on April 24th, 1902, and found that both ovaries were cystic and contained nodules of a spheroidal-celled carcinoma. Convalescence was uninterrupted, and she left the hospital on May 18th, 1902, her weight at that time being 10 st. 2 lbs. The patient suffered much pain in both breasts after the operation, but at the time of her discharge from the hospital the breasts were painless; the growth in the right breast was smaller and softer, whilst the nodules in the skin were much less marked than at the time of her admission. We saw her again on June 19th, when she weighed 11½ st. in her clothes, which led her to say that she had increased just 7 lbs. in weight. She reported herself as being greatly improved in health, and as having ceased to menstruate, although before the operation she had been perfectly regular. The left breast was nearly free from mastitis; the right was freely movable upon the underlying tissues, and the scar over it was much less nodular than it had been a month previously. Neither breast had undergone very much atrophy, and there was not much retraction of the nipple. The infra-clavicular glands had diminished in size, but had by no means disappeared. In July, 1902, I showed her again to my colleagues at consultations, and asked whether, in view of the improved conditions, it would not be desirable to remove the left breast and the axillary glands, as I feared that the presence of proliferating epithelial cells might increase the tendency to secondary deposits. They recommended me, however, to wait for some time longer, as the improvement was so marked and it seemed to be progressing. We saw the patient again on August 20th, and found that she was still in good health and with a greatly improved colour. The left breast was free from cancer. The patient complained of pain and swelling along the inner side of the right arm as low as the internal condyle of the humerus. Mr. Butlin, who happened to be present when she was being examined, kindly looked at the swelling and said he thought it was identical with the lymphatic œdema which is so often seen after extensive operations for cancer of the breast. The weight of the patient remained unchanged at 11 st. Three months ago she came to see us again, and her condition then appeared to be very similar to what you see at present, her weight still remaining the same. To-day she weighs 11 st. 4½ lbs., and all her symptoms are improved, whilst the cancer remains in abeyance.

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THE DIFFERENTIAL DIAGNOSIS AND TREATMENT OF CHRONIC DISEASES OF THE JOINTS.*

By ARTHUR P. LUFF, M.D., B.Sc., F.R.C.P.Lond.,

Physician in Charge of Out-Patients in St. Mary's Hospital.

LECTURE I.

THE correct diagnosis of chronic joint disease is often a matter of considerable difficulty, a difficulty which is not diminished by the general desire on the part of our patients to have a disease labelled with some distinctive title. There is much looseness in the diagnosis, and consequently in the treatment of joint affections, due to a great extent to the descriptions of joint diseases in text-books not being up to date with the advances of scientific knowledge as to their mode of origin and pathology. Perhaps the best classification of joint diseases is that of Bannatyne, who divides them into three great classes:—(1) the bacterial or toxic arthropathies; (2) the nerve arthropathies; and (3) the senile degenerative arthropathies. To this division I would add a fourth class, viz. those due to interference with the nutrition of the joints by circulatory changes, such as occur in Raynaud's disease. The term toxic is not meant to be confined to the products of bacterial activity only, but includes those arising from the tissues themselves, or by chemical processes, such as the toxic substance or substances causing gout. Another classification of arthritic cases is into (1) the essential arthropathies—*i. e.* those in which the joint troubles form the principal or prominent symptom of the disease, such as gout, rheumatism, rheumatoid arthritis, and senile arthritis; and (2) the accidental arthropathies—*i. e.* those which, although they may occur in the course of a disease, yet are not essential symptoms of that disease, such as gonor-

* Two lectures delivered at the Medical Graduates' College and Polyclinic on May 8th and 12th, 1903.

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rhœal, scarlatinal, malarial, tuberculous, syphilitic, pneumococcic, and nerve arthropathies. With regard to the bacterial arthropathies considerable discussion has arisen as to whether the bacteria or their products are the cause of the symptoms. I think, however, that the balance of evidence is strongly in favour of the joint troubles being due to the micro-organisms themselves, since the micro-organisms of rheumatism, gonorrhœa, pneumonia, and possibly rheumatoid arthritis have been found in the joint structures and joint fluids. Moreover, in those cases where operative treatment of the joints has been resorted to, the rapid subsidence of the symptoms points to the joints being the seat of bacterial infection, rather than being affected by toxic products elaborated elsewhere. Certainly it may be accepted that infection plays a most important part in the production of joint diseases, while a general defect of nutrition underlies many, if not all of the chronic forms of arthritis. Unfortunately, the term rheumatism has been applied with too much looseness and too generally. Now that it is known that acute rheumatism is itself an infective disease, it is recognised that many cases of arthritis which have been termed rheumatism certainly belong to the infective class.

I propose first to consider the differential diagnosis of the three forms of chronic joint disease that are most frequently met with, that are most apt to be confounded, and that therefore offer the principal difficulties in diagnosis, viz. gout, rheumatoid arthritis, and rheumatism. The differential diagnosis of these diseases is a very important matter, and it is all the more important to direct attention to such diagnosis, because undoubtedly in the past many cases of rheumatoid arthritis have escaped recognition, and have been diagnosed either as rheumatism or as gout. When it is considered that the treatment of these three diseases is quite different, the necessity of a correct diagnosis is manifest. I am sure that many cases which are diagnosed as chronic rheumatism of the joints are not chronic rheumatism at all; many of them are cases of rheumatoid arthritis, and a fair number are cases of gout. I believe chronic rheumatism of the joints—chronic arthritic rheumatism—to be a comparatively rare affection; at any rate, it is not very frequently met with. Cases are frequently diagnosed as cases of chronic rheumatism, in which there are great deformities of the joints, lipping of the cartilages, osteophytic outgrowths, and grating of the ends of the bones. Those are cases of rheumatoid arthritis. In chronic rheumatism, neither lipping of the cartilages nor the osteophytic outgrowths which are so diagnostic of rheumatoid arthritis ever occur.

Before proceeding to the question of differential diagnosis I must therefore ask your attention to a very brief consideration of those three types of disease.

Gout.

Gout I regard as a disease which is due to faulty metabolism, probably both intestinal and hepatic, as the result of which certain poisons (possibly the purin and other bodies, but of which we at present know but very little) are produced and lead to an auto-intoxication, which is an early factor in the development of the gouty condition. This auto-intoxication coincides with, or is followed by, in the majority of cases, a deposition of sodium biurate in certain of the joints or tissues, which constitutes the climax of the gouty attack. Certainly I think that with our increasing knowledge and experience, uric acid and its salts will in all probability have to be relegated to a position of subsidiary importance in the pathogenesis of gout. The joint manifestations are probably dependent upon much more general and much larger conditions than a mere excess of uric acid in the blood. The deposition of sodium biurate is possibly merely the sign of the disease, not the essence of it.

The appearances presented by the joints in cases of chronic gout are too well known to require any detailed description in these lectures. The joints of the hands are particularly apt to be gradually thickened, especially the metacarpo-phalangeal joints; and in addition there is a marked tendency for the gouty deposit to invade the fibrous tissues in the intervals between the joints, so that the trouble in many gouty cases is not limited to the joints. In marked cases the hand loses all its natural form, and has been compared to a bunch of carrots or of bananas.

Rheumatoid Arthritis.

The term "rheumatoid arthritis" is objectionable, as suggesting a causal connection with rheumatism. If, however, it is employed and understood

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as merely meaning an arthritis somewhat resembling some forms of rheumatism, the term may be retained, although the name "arthritis deformans" is less open to objection. I propose, however, in these lectures to retain the term "rheumatoid arthritis," owing to its long usage, as I am afraid that the description of that disease under another and less recognised name may lead to confusion. By rheumatoid arthritis I mean the disease known as "arthritis deformans," or "polyarthritis deformans," or "rheumatic gout." I think that very often the unfortunate name of "rheumatic gout," as applied to rheumatoid arthritis, is a cause of the two conditions being confused. Certainly it is a name which covers a multitude of sins and errors in diagnosis, and it is a term which ought to be abandoned. I cannot too forcibly impress upon you the fact that "rheumatic gout" possesses no entity as a separate disease.

The cases hitherto grouped together as rheumatoid arthritis undoubtedly include more than one disease. I should define true rheumatoid arthritis as a progressive degeneration of the joints, consisting of changes in the synovial membranes, cartilages, and bones, accompanied by atrophy of some structures and by hypertrophy of others. In chronic cases marked osteophytic outgrowths are peculiar to this disease. Heberden, in 1804, was one of the first to distinguish between this disease and rheumatism. He pointed out that there was swelling of the affected joint, but little or no fever, no great pain, and no redness of the skin; that the disease generally attacked joint after joint, and that it was very crippling; that the fingers and wrists were especially liable to the disease, and that the terminal phalangeal joints of the fingers were liable to become affected with nodosities, which have since become known as "Heberden's nodes." The disease occurs in acute, subacute, and chronic forms. The chronic form may be chronic from the first, or may be secondary to an acute or subacute attack. The acute and subacute forms are characterised by inflammatory changes in the affected joints, by erosion of cartilages and bones, by nerve and trophic phenomena, and by glandular enlargement. It is polyarticular, and in its acute and subacute forms occurs especially in children and young adults. The disease usually commences in one joint, commonly one of the metacarpo-phalangeal articulations, and then rapidly spreads to most of the other joints. The symmetrical nature of the affection is usually well marked, and the joints are painful, hot, and present a spindle-shaped enlargement, but no outgrowth or thickening of either cartilage or bone during the acute stage. The chronic forms are characterised by progressive thickening and hardening of all the joint structures, by the formation of osteophytes, by the lipping of cartilages, and by the development of deformities. The disease may affect several joints, or be confined to one or two. It most commonly occurs in middle life, and in females. Comparatively small injuries of a joint, especially of a small joint, may lead to rheumatoid arthritis, and to an extension of the process to other joints in a symmetrical order. The injuries are frequently the outcome of excessive work and strain, especially in elderly and enfeebled persons with a diminished power of resistance, increasing with years, and with imperfect nutrition.

The three divisions of the disease proposed by Charcot constitute the generally adopted classification of the forms of rheumatoid arthritis for a study of their symptoms. They are (1) cases with Heberden's nodes; (2) the general progressive form; and (3) the partial or monarticular form. This last-mentioned monarticular form is, however, in my opinion, an absolutely distinct disease from rheumatoid arthritis, and I shall describe it later among the senile arthropathies.

1. *Cases with Heberden's nodes.*—These cases represent the mildest degree of the disease. The nodes consist of little hard swellings of the finger joints, affecting almost entirely the terminal phalangeal, and are due to a very chronic form of rheumatoid arthritis. This type is more commonly met with in women than in men, and usually at or after the middle period of life. The nodules are due to enlargement of the ends of the bones, which are frequently covered by a pouch of the projecting synovial membrane, which acts somewhat as a bursa. The joints become swollen and tender. The cartilages are softened, and the ends of the bones are eburnated. The enlargements are osseous in character, but there may be a certain amount of increase of the periarticular fibrous tissues. After a time the disease usually becomes arrested, but the swellings remain, and eventually may cause no discomfort. Treatment cannot produce any dimi-

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nution in size of the bony growths, but may effect a decrease in size of the periarticular tissues referred to.

Heberden's nodes are frequently associated with some uterine disturbance. Heberden, in his original paper, particularly noted the fact that the thirty-three cases he described occurred only in women, and in women of middle age. This form of rheumatoid arthritis touches a point of age beyond which the influence of the sexual system is likely to be much diminished, and there is undoubtedly a direct connection between it and uterine troubles associated with the climacteric. The affection is not commonly very progressive, and never reaches to much deformity; but, on the other hand, there is no retrocession of the chronic arthritis. The uterine troubles and the active affection of the joints subside together.

Apart from Heberden's nodes, other forms of chronic arthritis are frequently associated with and determined by uterine and ovarian troubles. It has for some time been held that the joint troubles are due to reflex trophic influence, the excessive uterine irritation being reflected from the spinal cord to the joints; but I cannot help thinking that a more probable explanation is that the diseased uterus becomes the channel by which an infective or septic arthritis, generally leucorrhœal in origin, is started in the joints. In a similar way a chronic urethral and prostatic affection in men, quite apart from gonorrhœa, may give rise to a chronic arthritis; such an arthritis may also be started by the use of an imperfectly clean catheter. I have had under me in the course of the past twelve years six cases of severe chronic arthritis, indististinguishable from rheumatoid arthritis, in which the disease followed the removal of both ovaries. Whether in these cases the disease occurred in consequence of the removal of the ovaries, or in association with the diseases requiring their removal, I am unable to say.

2. *The general progressive form.*—Of this there are two varieties—the acute and chronic. The acute form has been previously referred to (see p. 387). It may resemble, and certainly has been mistaken for, acute articular rheumatism. It generally starts in one joint, and subsequently involves many. There is not much redness of the affected joints, and only moderate fever. It is most common in young adults and young women. Among the last mentioned it is often connected with recent delivery, rapid child-bearing, or excessive lactation. Rheumatoid arthritis certainly occurs in children, but very rarely, and even more rarely than the joint disease described by Still, to which I shall presently refer.

The chronic form is much commoner than the acute. The joints that have been most especially and actively used, according to the former occupation or employment of the patient, are those which usually show the first signs of the disease. The affection commences with slight swelling and pain on movement. The amount of effusion into the joint is variable, and may be marked or slight. The hands and feet, especially the hands, are most liable to be first affected, and the disease then tends to advance more or less up the limbs towards the trunk, obeying, as Charcot described, "the centripetal law." In extreme cases every joint in the body may be affected. The temporo-maxillary articulation becomes the seat of rheumatoid arthritis in about 25 per cent. of the total number of cases.

At a later period the articulations of the spine may become involved. The disease usually attacks the cervical vertebræ first, causing pain at the back of the neck, and rendering fixation of the neck and rotation of the head difficult. The dorsal and lumbar vertebræ may be next affected, so that in bad cases the spine may be converted into a rigid column. Pain may be very severe, especially at night, while, on the other hand, the case may proceed to extreme deformity without pain.

Very considerable alterations in the shape of the joints may occur from the formation of osteophytes, thickening of the capsules, and retraction of muscles. The cartilages become worn away at the centres, and the ends of the bones become eburnated by attrition and chronic osteitis. In such joints grating is readily obtained by rubbing the eburnated ends of the bones against each other. The locking of the joints, which sometimes ultimately occurs, is not due to true ankylosis, but to the presence of the projecting osteophytes, and to the thickening of the capsules of the joints. True ankylosis only occurs in the spinal column in cases of rheumatoid arthritis. Atrophy of the muscles from disuse is present in bad cases, with contractures tending to flex the thigh or to bend the knee or elbow. There is usually some increase of myotatic irritability, as shown by some exaggeration,

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frequently but slight, of the knee-jerks. Most patients finally reach a stage in which the disease becomes arrested, so that they are free from pain, and only are troubled with the associated crippling and consequent inconvenience.

Rheumatoid arthritis is nearly always associated with a certain amount of anæmia, the patients presenting a sallow appearance. Increased rapidity of the heart's action is a not uncommon accompaniment of the disease in its earlier stages, and cold and moist hands and feet are commonly met with. Subcutaneous fibroid nodules and periosteal nodes are occasionally present, especially in those cases which are secondary to rheumatism; and a rheumatoid pigmentation of the skin, somewhat resembling freckles in appearance, is not unfrequently seen, but is especially present in the acute stage. In a small proportion of cases a neuritis is present, but it probably is always secondary to the arthritis, and may be caused, as suggested by Bannatyne, either by the existing joint inflammatory process, or by the action of toxins circulating in the blood. Spender describes the following collateral symptoms, one or more of which are commonly present, as aids to diagnosis in doubtful cases:—(1) tachycardia; (2) pigmentation of the face, and perhaps numerous spots or stains on the arms; (3) cold and moist hands; (4) neuralgic twinges in the upper and lower limbs; (5) persistent neuralgic pain over the ball of the thumb and on the ulnar side of the wrist.

Rheumatism.

Rheumatism is a disorder which generally manifests itself as acute rheumatism or rheumatic fever; but in the chronic condition, although it generally manifests itself as an arthritic disorder—that is to say, a disorder affecting the joints, it may not manifest itself that way at all; it may simply show itself by the production of chorea, or by the production of erythema, or by the production of fibrous nodules, or by the production of endocarditis or pericarditis. Therefore rheumatism is a disorder which does not necessarily show itself as a joint affection; it may become manifest in some other way. In other words, there is the articular chronic rheumatism, and there is the abarticular chronic rheumatism—such as the choreic and erythematous forms. As an assistance in the diagnosis of a chronic articular affection, there is a rough but fairly sure test which is frequently of assistance in diagnosis: it is treatment with salicylate of soda. If the case responds well to this treatment, it is a case of rheumatism. If it does not respond to this treatment, the existence of rheumatoid arthritis or gout is fairly certain, as neither of these affections responds well to salicylates. When I hear the remark about "an obstinate case of rheumatism which has not done well with salicylates," I feel fairly sure that it is a case of gout or of rheumatoid arthritis—more probably the latter. Very many of the cases diagnosed as chronic rheumatism are cases of rheumatoid arthritis, or gout, or some form of infective arthritis or arthropathy. Though much less frequent than is generally supposed, chronic rheumatic affections of the joints do occur, but they never produce that permanent deformity which the other affections may, especially the lipping of the cartilages and the bony outgrowths already referred to. These chronic rheumatic affections of the joints are generally accompanied by the formation of fibrous nodules over the tendons, by the development of extra-articular fibrous thickening by the presence of subcutaneous fibres, fibrous nodules (especially on the fingers, elbows, and head), and not unfrequently by the indication of valvular disease of the heart. There is generally a well-marked history of rheumatism, and the disease is identical with that described as "chronic fibrous rheumatism." It must not be considered that the subcutaneous fibrous nodules occur only in connection with rheumatism. They are fairly frequently seen in cases of rheumatoid arthritis, and less frequently in gout, syphilis, influenza, and other diseases.

It should be carefully remembered that chronic rheumatism is not associated with the formation of bony or cartilaginous deposits in the joints. It shows little symmetry, and is usually associated with rheumatic pains shifting about from one place to another. It never affects the temporo-maxillary joints. As regards the actual diagnosis of rheumatism, it is usually fairly easy. If a patient complains of pains in the joints, which pain flies about from joint to joint, and generally affects some of the muscles at the same time, and if, in connection with these flying pains, there are indications of the presence of the rheumatic erythema—erythema

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nodosum—then the diagnosis of rheumatism is obvious. As a rule, the fitful way in which the joints are affected, the fairly rapid subsidence of the swellings of the joints, and the association of muscular pains, make the diagnosis a simple matter. Then the response of the disease to treatment by salicylates will settle the diagnosis. The absence of bony thickening and of bony grating distinguishes the disease from rheumatoid arthritis, and in addition the finger-joints are much less frequently affected.

I will now consider the differentiation of gout and rheumatoid arthritis, the two chronic joint diseases which are most frequently confounded.

The following distinguishing characters show how very different the two diseases are. In the first place, rheumatoid arthritis occurs most commonly in females; gout occurs mostly in males. Rheumatoid arthritis occurs more commonly amongst the poor and ill-nourished; gout mostly among the well-to-do and well-nourished. Rheumatoid arthritis is a disease which is improved by good dieting; in the case of a gouty person a spare and plain diet is indicated. The onset of rheumatoid arthritis is insidious; that of gout sudden and obvious. As regards the commencement of the attack, gout most commonly begins in one of the feet, especially in the great toe joint; rheumatoid arthritis, although ultimately it frequently affects many joints of both hands, nearly always begins in one joint, most commonly selecting one of the joints of the thumb, either carpo-metacarpal or metacarpo-phalangeal joint, after which it rapidly spreads to the other joints. Then as regards the appearance: in the case of rheumatoid arthritis there is no obvious swelling at first, and no marked redness. In the case of gout, at its commencement there is very obvious swelling, marked redness, and a shiny condition of the skin around the affected joint.

In rheumatoid arthritis there is very little pain at first. There is some aching in the joint, but it starts in a very insidious manner. It is this insidious character of the disease which is one of its bad features, for the patients do not seek advice until the affection is fairly advanced. Gout, however, begins in the most marked manner with severe pain, the patient as a rule waking up in the early morning with excruciating pain in the great toe. Therefore, if there is doubt as to whether a particular case is one of rheumatoid arthritis or of gout, the patient should be questioned as to the commencement of the attack, in order to ascertain whether it began with an obvious outburst of pain, and with swelling and redness of the joint, or whether it began very insidiously. Then as to the joint affections; apart from what has been stated as to gout generally beginning in the foot, and rheumatoid arthritis generally in the hand, there is one joint commonly affected in rheumatoid arthritis which is not affected in cases of gout, that is the temporo-maxillary articulation. I have never seen a case of gout in which the temporo-maxillary joint has been affected, whereas in rheumatoid arthritis it is fairly common for that joint to be affected.

In rheumatoid arthritis there is a special liability to the affection of the joints of the cervical vertebræ, as evidenced by pain and stiffness at the back of the neck. This is a most useful distinguishing sign. Another distinction is this, and it is perhaps one of the most important distinctions, that in connection with rheumatoid arthritis there is a remarkable symmetry in the affection of the smaller joints of the hands. In gout that symmetry is wanting. It was this symmetrical affection of the joints which led to the idea—which I believe to be absolutely erroneous—that rheumatoid arthritis is a nervous disease. Lastly, in a case of rheumatoid arthritis, sodium biurate is neither found in the joints nor in the blood, whereas in the gouty person sodium biurate exists both in the joints and in the blood.

It is not common to get rheumatoid arthritis and gout associated in the same individual. Undoubtedly rheumatoid arthritis may supervene in joints which have been the seat of any acute infective arthritis. Any affection which impairs the nutrition or weakens the resistance of joints offers a suitable condition for the development of rheumatoid arthritis. Hence rheumatoid arthritis may follow such diseases of the joints as acute rheumatism, gout, gonorrhœal arthritis, etc. This is the only sense in which such diseases are related to rheumatoid arthritis, in that they have by impairment of the joint structures so lowered the vitality as to render the joints more liable to the invasion of the micro-organism or toxin of rheumatoid arthritis. On the other hand, a person suffering from rheumatoid arthritis, who indulges in rich living for a lengthened period of time, and

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especially if he takes much wine, may develop gout, and so gouty deposits in the joints of a patient suffering from rheumatoid arthritis may occasionally be met with. This, however, is only a complication; there is no actual relationship between the two conditions, and one disease does not, in the strict sense of the term, predispose to the other.

A Form of Chronic Joint Disease in Children described by Still.

This is a disease closely resembling the rheumatoid arthritis of adults, and which until recently was always considered to be identical with rheumatoid arthritis, but which, as pointed out by Still, presents such marked differences as to suggest that it has a distinct pathology.

The disease consists of a chronic progressive enlargement of joints associated with enlargement of glands and enlargement of the spleen. The onset, which is usually insidious, almost always occurs before the second dentition, and girls are more commonly affected than boys. The enlargement of the joints is smooth and fusiform, and feels like a general thickening of the tissues around the joint, with none of the bony irregularities of the rheumatoid arthritis of adults. There is no redness, and as a rule no tenderness of the joints; and although creaking is frequently present, there is never any of the bony grating so commonly present in true rheumatoid arthritis, since in this disease there is a complete absence of the cartilaginous changes so characteristic of the latter disease. Limitation of movement, chiefly of extension, is almost always present. There is an absence of the extensive deformities of the hands so frequently present in the rheumatoid arthritis of adults. The affection is symmetrical. The joints earliest affected are usually the knees, wrists, and those of the cervical spine; the ankles, elbows, and fingers are subsequently affected. There is no tendency to suppuration nor to bony ankylosis. One of the most distinctive features is the affection of the lymphatic glands. The enlargement is general, but affects primarily and chiefly those related to the joints affected. The enlargement of the spleen is roughly proportionate to that of the glands, but does not extend to more than one half to one inch below the costal margin. The heart shows no evidence of valvular disease. Sweating is often profuse, and a certain amount of pyrexia, generally recurrent in character, may be present. The disease is usually accompanied by a general arrest of bodily development. The disease is not in itself fatal, but, after running a slow course, the joint disease becomes permanently stationary.

The four important points in the differential diagnosis of this disease from rheumatoid arthritis are (1) the enlargement of the glands; (2) the enlargement of the spleen; (3) the peculiar appearance and feel of the joints, with the absence of bony grating on manipulation, and the absence of osteophytic outgrowths; and (4) the fact that the disease begins nearly always in the knees or wrists, and affects the fingers much later, whereas rheumatoid arthritis in adults affects the small joints of the hands early, and frequently commences in them.

Senile Arthropathies.

Senile arthritis in the great majority of cases affects only one joint, usually the hip or shoulder. Occasionally it occurs in the spinal column. This is the form of arthritis frequently known as the monarticular form of rheumatoid arthritis, but it should be regarded as a local degeneration, progressive in its nature. It is, I believe, an entirely different disease from rheumatoid arthritis, and is probably not microbic in origin. In the hip the disease is known as "morbus coxæ senilis," and in the spine it is known as "spondylitis deformans." It occurs in elderly people, and is in the majority of cases started by some local injury, and is common in old persons who have had to carry heavy weights, or who make excessive use of their lower limbs in the course of their occupation. The arthritis is persistent, and, if not suitably treated, progressive. Grating is nearly always present, with muscular wasting, shortening of the limb, and limitation of movement accompanied by pain. On deep pressure over the affected joint pain is always elicited. The synovial membrane of the joint shows chronic inflammatory changes, along with destruction of cartilages and softening of the articular ends of the bones. The last-mentioned condition is in marked contrast to the eburnation of the ends of the bones in cases of rheumatoid arthritis.

Gonorrhœal Arthritis.

This form of arthritis is also known as "gonorrhœal rheumatism," a term which should

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be abandoned as being misleading in its character. It is always secondary to a gonorrhœal attack elsewhere. This attack is generally in the urethra, but is not necessarily so, as some cases have followed infection of the conjunctiva. The arthritis is due to the presence of the gonococcus in the joints. It usually appears at the decline of the urethritis, or even when this appears to be cured. It is not, therefore, directly proportional to the acuteness of the urethritis. It rarely develops before the third week, and its advent may be postponed for several months after the onset of the gonorrhœa. Time is required for the gonococci to reach the glandular tissues of the prostate, whence they are enabled to enter the circulation. This form of arthritis is most liable to affect the larger joints, especially the knees; the joints that may also be affected are the ankles, elbows, small joints of the feet, shoulders, wrists, small joints of the hands, and the sterno-clavicular joints.

The disease is apt to be mistaken for subacute rheumatism, but it may be differentiated by the comparative absence of temperature, and by the non-reaction of the affection to salicylates. As a rule the diagnosis is rendered easy by the presence of a gonorrhœal discharge, but this is apt to be overlooked in women, on account of the frequency of a leucorrhœal discharge.

The actual condition of gonorrhœal arthritis may be one of synovial effusion, or it may be limited to thickening of the joint capsule and of the peri-articular tissues. If neglected suppuration may occur in the joint.

A form of infective arthritis which is somewhat akin to gonorrhœal arthritis is that accompanying ophthalmia neonatorum. Rapid recovery always follows on fixation of the affected limb and local treatment of the ophthalmia. Two other forms of septic or infective arthritis somewhat resembling gonorrhœal arthritis are those which occasionally occur in connection with profuse leucorrhœal discharges, and in connection with bronchiectasis.

Arthropathies due to Infective Fevers.

In these lectures I am dealing only with the chronic forms of arthritis, but it is necessary to briefly refer to the forms of arthritis which may occur in connection with any of the infective fevers. As generally seen, these are forms of acute arthritis, but occasionally they persist for a long time, and so become cases of chronic arthritis. These forms of arthritis have been known to occur in connection with scarlet fever, pneumonia, malaria, septicæmia, pyæmia, enteric fever, Maltese fever, measles, influenza, dysentery, glanders, erysipelas, etc. It is, however, only within the last few years that the joint troubles that occasionally occur in the course of the specific diseases have come to be regarded as due to the micro-organisms or poisons of the respective diseases. Formerly they were looked on as manifestations of true rheumatism occurring during the course of the disease. All these forms of arthritis are especially liable to occur in a joint or joints which have been previously damaged, either by disease or by mechanical injury.

Scarlatinal arthritis.—This is, perhaps, the best known of the type of arthritis met with in the various infective diseases. The elbows and knees are the joints most frequently affected; the smaller joints of the upper extremities follow next.

Pneumococcic arthritis.—The occurrence of arthritis during an attack of pneumonia has long been known, but until comparatively recently it was generally ascribed to a concurrent attack of rheumatism, and was not directly connected with the attack of pneumonia. A strong tendency to ankylosis is one of the special characteristics of an infective arthritis. Previous damage to a joint, as by injury, rheumatism, or gout, favours the localisation of pneumococcic arthritis. The influence of injuries in predisposing joints to attacks of infective or septic arthritis should be carefully borne in mind, as otherwise cases which are really those of infective arthritis may be considered simply traumatic in origin, and so the possibly necessary operative treatment of the joint may never be considered.

Malarial arthritis. — The parasite, although different in its life history from a bacterium, yet from the point of view of causation of arthritis may be considered as such. Malarial arthritis due to the parasite is a comparatively rare affection, but rheumatoid arthritis coincident with a malarial attack is not by any means uncommon.

Quiet Effusion into Knee-joints.

This affection was first described by Sir William H. Bennett, and was stated by him to occur only in girls and women, and to be always associated with menstrual irregularity or uterine trouble. It occurs mainly at the time of puberty, and at the

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climacteric. A similar condition has, however, been observed in males. The affection consists of a passive effusion into the joint, and rarely occurs in any other joint than the knee. It is unattended by pain, and a large number of the patients are unaware of its existence, unless attention is called to it accidentally. It is best recognised when the patient is standing in the upright position, when the fluid sinks to the lower part of the joint cavity, and sometimes forms a pouch-like overhanging of the synovial membrane at its lower anterior aspect. Attention is generally called to the condition by a slight injury, such as a twist or fall. The joints of the two sides are usually involved at the same time, but the effusion is, as a rule, much more marked on one side than on the other, that on the right side being generally the greater.

Nerve Degenerative Arthropathies.

These are joint affections that occur in tabes, ataxy, paraplegia, progressive muscular atrophy, etc. The joint troubles are due to abnormal trophic influences started by affections of the central nerve organs, as the result of which inflammatory troubles, generally of a very chronic nature, are set up, characterised by alterations in the ends of the bones, ulceration of cartilages, and formation of fibrous adhesions. Of all the chronic joint affections these are perhaps the easiest of diagnosis, on account of their association with an easily recognisable disease of the nervous system of a degenerative nature.

Tuberculous Disease.

The history of the case, both family and personal, is of great assistance in making a diagnosis in this form of joint disease. The swelling is frequently spindle-shaped. When the disease is advanced the joint is in a condition of organised fibrous tuberculosis, and consequently nothing of diagnostic value is to be made out by radiographs.

Syphilitic Joint Disease.

This is commonly chronic. There is effusion with thickening of the capsules of the medium-sized and larger joints, with occasional bony thickening and lipping of the smaller joints, which may at times closely simulate rheumatoid arthritis. The presence of a gumma, and of old iritis and other indications of syphilis, will frequently assist in making a correct diagnosis. A form of joint disease occurring in the later stages of syphilis has been described by Whitfield, which presents many resemblances to a joint recovering from acute gout. The joint is considerably swollen, the surface shiny, the skin being of a dark red colour, with distended veins crossing over the joint. As a rule it is exquisitely tender to the touch. The diagnosis is, as a rule, rendered easy by the discovery of a periosteal gumma over one of the bones. The joint disease itself is undoubtedly gummatous in nature, the gummatous infiltration starting in the ligaments and periarticular tissues.

Pulmonary Osteo-arthropathy.

This is a rare disease. It is secondary to some chronic pulmonary disease, such as phthisis, empyema, and chronic bronchitis. The disease may possibly be tuberculous, or due to some toxic agent. It is characterised by considerable enlargement of the hands, wrists, feet, and ankles. It is apt to be mistaken for acromegaly, but may be distinguished by the clubbing of the finger ends and the enlargement of the wrists, which conditions are absent in acromegaly.

Joint Changes in Raynaud's Disease.

The joints are much thickened; they present little indication of fluid distension of the cavities; there is a good deal of thickening of the bone, and considerable pain and tenderness. The condition is apt to be mistaken for rheumatoid arthritis, but the diagnosis is rendered easy by the association with the circulatory changes accompanying Raynaud's disease.

Typhoid Perforation.—Voorsanger lays stress on the white blood count in the diagnosis of typhoid, attaching as much, if not more, importance to it than to the Widal reaction. The leukopenia occurs during the first few days, and is always very positive, being, as a general rule, between 3000 and 5000. A rise always means a complication. In a case of perforation he describes the only symptoms were the dropping temperature, the high pulse, and tympanites.—*Journ. A.M.A.*, Sept. 5th, 1903.

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A CLINICAL LECTURE
ON
SCOLIOSIS.

Delivered at St. Bartholomew's Hospital.
By W. BRUCE CLARKE, M.A., M.B., F.R.C.S.,
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You will remember, gentlemen, that on a former occasion when we met in this theatre we were discussing the subject of flat-foot, and I pointed out to you that there were two definite conditions by which flat-foot was very largely influenced. First, there were certain nutritional changes in the tissues themselves; and secondly, we had to reckon with the effect of the ordinary forces of gravity and with mechanical forces in the production of that deformity which we described as flat-foot. In other words, and putting it into a nutshell, there is first to be considered the nature of the organism, and secondly the nature of the conditions under which that organism was placed.

I think when we come to speak about crooked spines we shall find the same principle generally prevails. There is first of all the nature of the organism or individual to be considered—that is to say, the individual in whom these deformities take place,—and then there are the sort of influences to which that individual is subjected. I think if you look into the statements of many of the books, and particularly the older text-books, you will agree that the tendency is to magnify the mechanical conditions; whereas perhaps the tendency to-day is more to magnify those conditions which we may describe as nutritional. But I want to give you as fair an idea to-day as I can of how much can be put down to one set of forces, and how much to the other. I am of course now looking at the matter purely from a clinical point of view. But before I begin to speak of curvature of the spine I will briefly give you a list of the cases which we must bear in mind when we are talking about the subject. First of all there is *Pott's disease.* Secondly there is what we may describe as an ordinary *mechanical curvature,*—that is to say, there are those curvatures which we are quite familiar with, which undoubtedly do exist, however rare they are, dependent upon certain conditions which we may describe as mechanical. And lastly, there is of course that large division which we describe as *lateral.* Some people will tell you there are only two forms of curvature of the spine—one angular curvature, and the other is lateral curvature. But I hope to be able to convince you to-day that the class which is described as mechanical is a *bonâ fide* class, and must be placed quite in a separate category. I shall not say much about any of these diseases, except with the view of diagnosis. I shall assume that you all know what is intended to be conveyed by the term Pott's disease. You know that it depends upon tubercular disease of the vertebral column, and produces a well-marked angular curvature. But it is not easy in some instances to be absolutely certain whether you are dealing with Pott's curvature or with curvature dependent on some other cause, when you are called upon to diagnose these cases in very early stages. That is a very important point to bear in mind. Here I have the skeleton of a well-marked case of angular curvature and there are plenty of cases about; you can see them outside walking about in the flesh, and there are plenty upstairs in the museum in spirit. You can easily find out how to distinguish the most obvious cases of Pott's disease.

Having said this much about Pott's disease, let me refer you to those conditions some of which, at any rate, are dependent upon mechanical causes. First of all let us take the ordinary curved spine of an elderly individual. Although it is not to be regarded as a necessary incident of old age, yet many people who live to old age become decidedly shorter and more bent in the spine. I have here a spine which in all probability belonged to an aged individual. Will you notice, if you please, that the curvature is an absolutely uniform one? That is to say, it may be in the best sense of the term described as postural. In other words, it has been produced presumably by a person who probably spent much of his time sitting in a chair and gradually leaning forward. If you look at a spine of this description you will find that the changes which occur in the spine are totally different from those which occur in a well-marked case of lateral curvature, or scoliosis. To begin with, if you look at these vertebræ, few if any of them are more wedge-shaped than they should be; most of the deformity has been produced at the expense of the intervertebral discs, and to a comparatively small

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extent at the expense of the vertebræ. If the bones are affected as well, the disease is generally spoken of as rheumatic, and in many instances is rheumatic, though I do not think we are always correct in calling it so. It occurs at the margins of the vertebræ. You have a certain number of outgrowths at the margins of the vertebræ which perhaps must be put down to some such disease as rheumatism; at any rate this spine has suffered from a form of disease which impedes the movements of the vertebral column and gives rise to that immobility with which we are acquainted as age advances. And bear in mind that we have none of the ordinary deformities of the spine which you meet with in well-marked cases of scoliosis.

What other kinds of spine are there in which we get none of those well-marked deformities which I shall speak about directly? I will tell you. There is the curvature of an empyema. You have probably all read of cases in which empyema has destroyed one lung, and in which the patient is bent to one side because the opposite lung has enlarged and taken the place of the lung which has been diseased and destroyed. I do not think there is any specimen in our museum which undoubtedly has been caused by a condition of that kind. But I do not think anyone, even the most sceptical, would have any doubt in his mind that such a condition of things occurs; and I expect many of you have seen a condition of this sort existing during life. If we do get an opportunity for a post-mortem on these cases some years after the empyema has occurred, what do we find? We find that the spine is bent over, and to a certain extent laterally curved; but we get no rotation of the vertebræ one upon another, or any of the ordinary changes which we meet with in cases of lateral scoliosis. Let us take another case, in which certainly I think we must admit that the deformity is obviously mechanical—for example, where one leg is shorter than its fellow. It is known to all of us that in such a condition you get a certain amount of lateral curvature of the spine. But as a rule you can correct that lateral curvature of the spine without the smallest difficulty by means of a high boot, at any rate, if you get it at an early stage, and you do not get any of this marked rotation about the spine which we see in the specimen on the table. As a rule it is a curvature which occurs in the lumbar spine, just as I may temporarily have a curvature in my lumbar spine because I am standing more on one leg than on the other—standing at ease, as it is called. That is a curvature which is only temporary, and which can readily be corrected; and the reason, I suppose, that a curvature of that kind is always temporary is because the vertebræ are sufficiently strong to resist the extra temporary pressure which is put upon them. That brings me to another point which I want you to realise, that wherever changes in position are of long duration, there must be nutritional change in the bone itself; *i. e.* there must be or must have been some peculiarity of the organism, and not merely a mechanical condition to which that organism is subjected. In my last lecture I pointed out to you the connection between rheumatism and flat-foot, and that a very large number of the cases which we meet with are cases in which we can not only trace a history of rheumatism, but in which, if we can cure the patient of his rheumatism, we can often cure him of his flat-foot. Since I delivered my last lecture I happen to have had a patient in the wards in whom the relation between rheumatism and flat-foot changes was even more obvious than it was in any of the cases I was able to refer to on that occasion. In this particular instance the effect of the bones being softened by an attack of rheumatism was such that the patient had his foot, so to speak, turned inside out; at any rate, what happened was that instead of the foot being arched upwards, as it should be, it was buckled downwards, and the result was that he had got a hollow on the top of the dorsum of his foot into which one could put two fingers of one's hand; it was a depression very much greater than you would expect to find in any ordinary person's foot in the sole. I could only correct it by dividing the tendons on the dorsum of the foot, the tibialis anticus, and the extensor tendons, and gradually righting the foot again, which it was easy to do under an anæsthetic. Whether it will remain permanently in its proper position is another thing. But I want you to realise that, given a softened foot, then any mechanical condition which acts upon that foot produces a deformity. Precisely the same thing happens in the back.

Now let us refer to one or two other conditions in which not only are there mechanical causes, but

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in which there obviously are nutritional changes at work as well. You may say that in the bent spine of old age which I have before me there are obviously nutritional changes at work as well. Possibly you may be right; these bones may be softened; but there is no certain evidence that it is so. But in this other specimen which I have here there is very definite evidence that the bones themselves were softened. This is the spine of a case of osteitis deformans. Some of you are probably familiar with the pictures of patients suffering from well-marked osteitis deformans—people who look in a sort of decrepid condition, with their hands hanging down nearly to their knees. Their spines are considerably bent, and not only are their bones altered in condition, but they are to a certain extent deprived of their phosphates. In these cases you have a softened condition of bone, and in addition you have a mechanical condition which acts upon those bones—namely, the weight of the body and the question of posture comes in as well, —and the result is a deformity of the spine such as you see here. They are the intermediate links between the cases of mechanical and lateral curvature properly so called, or scoliosis, about which I want to speak to-day. Afterwards I will pass to the question of what we can do to arrest, and, if possible, to cure these cases. If you want to know what the deformity is, you have only to take a specimen of this kind, or look at the people who are suffering from this condition when they are stripped to the waist in the orthopædic department. You will then fully realise what we mean by well-marked cases of scoliosis. The ordinary symptom which the patient, or his friends, complains of is this:—You are dealing perhaps with a growing child, and the parents say that one shoulder is growing out. In one boy you will find one shoulder is higher than the other. In another case the back sticks out in an uncomfortable way. In a girl perhaps it has been noticed that one breast is more prominent than the other; and so on. The friends generally notice that there is a great lack of symmetry in the chest or back; and when you come to investigate that lack of symmetry you will find that it depends as a rule on curvature of the spinal column. That curvature of the spinal column, when the patient is stripped, generally presents these peculiarities. There is perhaps a prominence below on one side and a prominence above on the other. Perhaps on one side there seems to be no waist where the waist ought to be, and on the other side the waist is unduly marked. When you look at the line of the spine you will find you can see the curve through the skin almost as easily as you can see it in the specimen which I am showing you. There are two curves, one to the right in one direction, and another to the left in the other. These are spoken of as compensatory curves. The question of a compensatory curve is very simple. The object of a compensatory curve is simply to enable the person to keep his head up straight. If I have a curve of my lumbar spine the tendency would be to throw my head over to one side. That would be so uncomfortable and inconvenient that Nature corrects that curve and gradually makes a compensatory curve to enable the head to balance itself on the trunk. In other words, it is a question of gravity. Therefore, if you have a curve in one direction you are certain to get a compensatory curve in the opposite direction. You must not imagine that you get a curve developed first in one direction, and that a compensatory curve is formed later. The two curves go on simultaneously and imperceptibly; a little bit of turning over of the spine in one direction is sufficient to warrant the spine in correcting itself by curving upwards in a contrary direction the moment the body is thrown into a position of discomfort. But this curve is something more than a compensatory curve; we shall find in specimens of this condition a certain amount of rotation of the spine as well; and if you draw a series of lines at right angles to the centre of the bodies of the vertebræ, you will not find your lines will lie vertically one over the other; they will tend to arrange themselves spirally like the treads of a spiral staircase, and where the curve is reversed and the compensating curve occurs, these lines will reverse their positions too. Many explanations have been given of this rotation; but I do not think we can really understand what rotation of the vertebræ is due to unless we go further into the question and try to make out exactly in what part of the bodies of the vertebræ themselves this bending takes place. If we take an early specimen of this sort we shall find that where the changes are largely taking place is at the growing margin of the vertebra; in

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other words, it is at the junction between the body of the vertebra and its epiphysis. And if we want to see that these changes in the vertebral bodies are almost independent of mechanical causes, we have only to look at these two animals whose skeletons I have had brought down. In this calf and this fowl you will see it is possible to get lateral curvature produced in quadruped animals. Look particularly at the calf; no amount of pressure on the fore-legs could have produced this deformity. I do not say that that particular curve was not dependent to some extent on mechanical causes; obviously that is the case; but it is also obvious it was not determined solely by the weight of the body, as we might imagine if we looked at man alone. It has been assumed that in ourselves the weight of the body acting on the trunk would produce a curve of the description I show you here. I think a deformed animal of that description is at once an answer to that question, because it shows that deformities of that kind may be produced in quadruped animals in which the weight of the body acts in a totally different way upon the spine. In the case of an ordinary rickety child, let us first of all notice the symptoms which we find in connection with it. As a general rule we are told that at the ordinary rickety age the child will not sit upright, that it is very easily fatigued, and so on. We take that child and examine it carefully all over, and we find well-marked signs of rickets, such as enlargement of the ends of the bones, beading of the ribs. The belly is larger than normal and the head is more prominent than it should be, and we conclude that the child has a rickety spine. Why it is that rickets should have affected that child's spine, or concentrated its attention upon the spine, or upon particular bones, we do not know any more than we know why syphilis in one individual attacks one particular set of organs or structures, and in another a part of the osseous system to the exclusion of all other parts. Still there is no doubt about this, that in many children we do find this condition existing only in the spine. And what is more, we assume it is rickety in the first place because the bones, when they are analysed, present the ordinary characters of rickets; and secondly, because we find that the means which we can adopt for straightening ordinary rickety long bones are equally applicable in the case of the spine. If we apply those same principles to the spine, that is to say, if we take a child and insist upon it first of all being kept for a certain number of hours each day on its back—which is not easy to do for various reasons, and that is why many of these rickety spines are not cured,—we can be perfectly certain we are arresting the progress of that child's rickety spine. And if we can feed that child satisfactorily by means of any of the ordinary forms of food which are given in connection with rickets, we shall as certainly cure that child of rickety spine as we can cure a bent leg or beady ribs, or any other of the symptoms which occur in connection with rickets.

If, on the other hand, we leave that child alone, or if the friends do not see their way to have the child treated satisfactorily, and we see that child later in life, what will be that child's condition? We shall find that the deformity has increased. But even if it has remained stationary it will not be in all probability a single curve, such as you meet with in rickets to start with; but you will get a certain amount of lateral curve or rotation of the spine superadded to it. Here is a case which I do not think there can be any doubt was in the first instance rickets. There are all the evidences of softening of the pelvis, with the extreme deformity of that bone which we meet with in rickets; and, superadded to that, we have a certain amount of this scoliosis or rotation of vertebræ which is so characteristic of lateral curvature. Sometimes these changes in connection with rickety spines do not come to a standstill, but go on getting worse. We then get that condition of rickets in adolescence, or what is called rachitis adolescentium, which we have in recent years been taught to reckon with. I do not mean to say there are many of this sort of cases existing. But I think we may assume, almost as an axiom, that if we find from time to time well-marked cases of a given disease, it is certain there must be cases of very much slighter character which are very likely to go unrecognised. And the more we observe the more we discover these hitherto unrecognised cases from time to time, and get evidence of the fact that many are unrecognised now. Supposing a case of rickety spine of this description which I am showing you is examined, what do we find? I mean if it is examined pathologically, and not simply by looking at the individual himself, or making a superficial examination of the specimen. What we find is

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this:—the disease has centred itself upon that portion of the body which we know was existent at the time; in other words, the body itself, which was not thoroughly and completely ossified, has been to a great extent pressed. Whereas if we come to an ordinary case of lateral curvature, what we find is that the disease is concentrating itself on the junction of the epiphysis and diaphysis of these particular bones; and I think if we examine any considerable number of these cases we shall come to the conclusion that the cases of so-called lateral scoliosis with which we are dealing are just as certainly due to some sort of nutritional change of one kind or another in the vertebræ as those cases of rickety spine which we deal with in children. That is a very important point to have reached, because if you get into your head the idea that we are dealing with a definite disease of the individual when we are dealing with most of the well-marked cases of lateral curvature, you will perhaps be inclined to treat it from a better standpoint than if you look upon it merely as a deformed condition brought about by mechanical causes. Practically, when we are called upon to treat these cases we are obliged to look upon them as conditions kept up by mechanical causes, because, as a rule, we are not called upon to treat these cases in the early stages, when the nutritional changes are the main trouble from which the patient is suffering. We are called upon to treat the results of the deformities, and perhaps supply them with instruments, when the nutritional changes have passed away and have merely left behind them certain mechanical disabilities, which are rendered far worse by the awkward conditions under which the muscular tissues and bones have to act.

You will ask me,—what evidence is there for regarding these deformities as rickety, beyond the analogies I have just referred to? It is very difficult to answer this question. Let me try to do so by an illustration rather than an analogy. I think every one now admits there is what is called a pre-tubercular stage in many cases of tuberculosis; that is to say, you meet with persons who are weak and liable to catch tuberculosis, and when they are in this condition they are very prone to be attacked by the tubercle bacillus. And in so many instances has this condition been observed that people have got to pretty well accept it as an axiom that there is such a thing as a pre-tubercular stage; in other words, a stage before the tubercular germ takes root in the body and produces that condition which we know as tuberculosis. And I am sure it is equally true that there is a pre-curvature stage; in other words, there is a nutritional change in the first instance which produces this condition of scoliosis about which we are talking; and that afterwards there are the mechanical causes which, acting upon the softened bones, produce that condition which we call lateral curvature. The instance I shall lay before you is that of a child. The case, I think, is interesting from many points of view. The mother of this child was suffering, and had suffered for many years, from a certain amount of lateral curvature. When I first discovered it she said it had come on when she was somewhere about twelve or fourteen years of age, and that it had not been thought much of. She had been told, according to the fashion of the day, to lie down for a certain number of hours each day, and a number of other things were done for her. The result was she had grown up to adult life with a well-marked lateral curvature, which one could distinctly see when she was stripped. She was very intelligent, and one day she called my attention to the fact that her daughter, at about the same age as that at which she herself was attacked, who up to that time had been straight—because I had examined her spine, and so had her mother, and found it all right,—was getting into a weakly condition. And the mother volunteered to me the statement—and it was confirmed by a sister of hers; that is to say, an aunt of the child—that at the particular time when her own curvature began to develop she got into a weakly state herself. Her daughter also was growing rapidly, and was in a weakly state. There was nothing particularly wrong with the child; it was taking its food particularly well, but the time was coming when the onset of puberty might be expected, when a considerable amount of extra strain is thrown upon the constitution. You will say these are vague matters. Of course they are, but they are as exact as you can get from the clinical point of view, and I do not see how, in the present state of our knowledge, we can get much beyond that and look at the matter purely from an experimental point of view. To cut a long

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story short, the mother was exceedingly anxious, just as I was, that great care should be taken to prevent this child, if possible, from developing a curvature of the spine. Accordingly, under my supervision, for somewhere about two years we went through a course of treatment for this child. In the first place we took great care of the child's digestion—that it should only have such things as it could easily and readily assimilate. The appetite at these times is liable to be fitful. We also took care that the child should not get over-tired. What did we do to prevent it getting over-tired? The mother being an observant woman, I got her to ascertain when the child was tired, and take care that the child lay in a recumbent posture for a certain length of time each day. The amount of recumbency was given by the child's sensations. If the child complained that she was very tired and fatigued we gave more recumbency, but if she seemed to be able to sit up longer we allowed her to do so. We took care by careful watching that the child's spine did not get into a curved condition. We noticed when the patient sat up that it seemed to drop first to one side and then to the other; and by having the muscles thoroughly massaged, by giving plenty of open-air exercise, and so on, we managed to tide her over a couple of years. At the end of that time the child got brighter and better looking, and improved in general appearance; there was more colour in her cheeks. In other words, it became obvious to everybody that the child had taken a fresh start in life, and had got out of her weakly state of health and was getting stronger. With this improved condition seemed to disappear any tendency towards curvature of the spine. You may say, how do you know this child was going to develop curvature of the spine? I cannot answer this question any more than you can, but I say this, that when you find that an intelligent mother asserts, and is supported in the statement by an intelligent sister, that she went through a similar stage at that period of life herself, and she was not taken care of, and did develop curvature of the spine; and when you find her daughter enters upon a similar pathological condition, as far as you can judge, and we do take care of her, and that condition of curvature of the spine does not come on; I say there is a certain amount of evidence, and I think good evidence, to show that in all probability that child would have developed some sort of lateral curvature if she had been put under the conditions to do so. Of course, if one could get half a dozen or twenty or thirty cases carefully looked after, I think you would go a long way towards proving that nutritional changes are at the bottom of many of these lateral curvatures of the spine to which I am referring; and I think that that is a point which all of you when you get into practice may very well be on the look-out for. I am certain there are plenty of these cases about, and if people's eyes were opened to the subject they would find plenty of them, and would probaby be able to arrest their development.

You will say, are there no other more definite means that you can tell us to prevent these various curvatures of the spine? What about the question of muscular exercises? Well, no one puts a greater value upon muscular exercises than I do. But let me tell you at once that if you have got well-marked curvature of the spine, such as you see in this individual we have before us, you may order muscular exercises or any form of exercises you like to your heart's content, but you can never cure that spine and make it a straight spine, such as all those who are in this room presumably possess. You may make that person comfortable, you may make him able to go about ordinary work, you may make him so that he will not be able to see his spine is curved when fitted by a tailor or stay-maker; but you will never be able to give such a person a satisfactory straight spine again. But the large number of the cases which come to us and which we treat, and which we are liable to confound with lateral scoliosis, are cases of weakness of the muscles, which are sometimes spoken of as if they were the same thing as lateral scoliosis. I do not believe they are; some of them may be lateral scoliosis in an incipient stage, but the majority of them are cases of weakness of spine in which we can no doubt treat them very satisfactorily by muscular exercises of various kinds.

In the short time which remains at my disposal I will tell you briefly what those exercises are and the general principles—I cannot do more—on which one carries them out. Of course the assumption underlying all these exercises is that muscular tissue and balancing masses of muscle are necessary in order to keep the spine in a straight position. We may make too much

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of that statement, but broadly speaking the statement is correct. For example, if one of my erector spinæ muscles were suddenly to become destroyed from any cause the assumption is, and probably it would be correct, that I should naturally tumble over to the side on which I had got good erector spinæ, simply because the tendency would be for the erector spinæ on the healthy side to pull my spine over in that way, and, having no counterbalancing erector spinæ on the other side, I should not be able to balance myself. Therefore we shall find that what we try to do as far as possible is to develop symmetrical masses of muscle on the two sides of the vertebral column. How do we accomplish this? The main principle is this: we lay the patient down flat on a table or board or hard bed, and having got the legs fairly fixed automatically, or fixed if necessary by somebody else holding them lightly, we try as far as possible to develop the two halves of the muscular system on either side of the spinal column; and we do it in the following manner:—We stretch out the arms laterally, and press against the individual who is lying upon the table. In other words, we ask the individual who is on the table to hold his arms up above his head, and let the person who is standing by try to prevent him from performing that movement by pushing in the opposite direction. You may say that only acts upon the two arms. But that is not so; it acts upon the vertebral column. If you want to prove that, lay the patient upon his stomach and watch the action of the muscles, and you will find that the various masses of muscles on the sides of the vertebral column, from the glutei to the muscles which attach the head to the neck, will be brought into action by the manœuvres I have just described. These muscles can be seen to stand out when these manœuvres are being carried out. Of course you want an intelligent person to carry out these evolutions, and you want them in addition to notice whether any particular set of movements is weaker than the remainder, and to try if possible to specially correct these weak movements by means of muscular exercises directed towards that particular end. Those are the general principles on which these various movements are carried out. Of course it is impossible to do more in a lecture like this than to point out the principles upon which the thing is done. To familiarise yourself with the practice you must come to the orthopædic department and see how it is done. If you realise those principles, and if in addition you realise that in most of these cases of lateral curvature or scoliosis with which we are dealing you have a definite nutritional change to treat, and that if you get it in a sufficiently early stage you can combat it by general treatment, food, and drugs, I think you will agree with me that it has some analogies with that change which occurs in rickets in children. If you have got so far you will have surmounted the first stage of treatment. At any rate I hope I have made it clear to you that you are not merely to treat the purely mechanical condition, and that you fully realise that though you cannot make a crooked spine straight, you can do a great deal to prevent such a condition from occurring.

Synorchidia in Conservative Operations on the Testicles.—Pascale has experimented on animals and the results have been confirmed, demonstrating that it is possible to unite the two testicles and that a single vas deferens will suffice for both. This retains the gland and its internal secretion, and allows the latter to issue by the natural channels, while the parts most liable to be infected by a tuberculous process in the epididymis may be removed. His technique is the systematic resection of the entire tuberculous epididymis and vas deferens on the side affected, leaving the vessels and nerves intact. He then opens up the rete testis and determines the integrity of this part of the gland before proceeding further. This portion of the gland is then implanted on the part of the sound testicle opposite to it, which is very easily done after removing the intertesticular septum. In four cases treated in this manner the results have fully confirmed the successes on animals previously published. They establish that the functional activity of a testicle thus implanted on its mate is maintained even after complete extirpation of its vas and epididymis, retaining the vascular and nervous elements. He calls this union "synorchidia," and adds that the vessels in the rete testis of the corpus Highmorianum retain their normal current, as they open at the base of the spermatic lobules, and the current passes through them and the straight tubes to the corpus Highmorianum on the sound side.—*Journ. A.M.A.*, Sept. 5th, 1903.

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THE CLINICAL JOURNAL,

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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The Index and Title-page for Vol. XXII will be published with the next issue of THE CLINICAL JOURNAL.

A CLINICAL LECTURE

ON

TERMINAL PENILE PAIN,

With special reference to some Cases of Calculus Impacted in the Ureter.

Delivered at St. George's Hospital, May 19th, 1903,

By SIR WILLIAM BENNETT, K.C.V.O., F.R.C.S.,

Surgeon to the Hospital.

GENTLEMEN,—Some very interesting points occur in connection with the symptom of pain at the end of the penis. It is one of the cardinal signs generally said to be produced by stone or other conditions of disease in the bladder; there are, however, conditions outside the bladder which give rise to it, and as we have recently had a case which illustrates extremely well one of the causes of this pain occurring independently of bladder disease, it is a good opportunity for considering the matter, which is really a good practical question. There is a little boy upstairs in Fitz-William Ward, nine years of age, who has complained of pain in the stomach for some long time; we do not quite know how long, but at all events for several years. Latterly he has complained of pain not in the stomach, but at the end of his penis, so much so that he at times is found to be pulling the end of his penis, as children so often do who have stone in the bladder. On Good Friday, to make the case appear more like one of stone in the bladder, he passed a little blood in his urine. So far as is known he had never done this before, nor has he done so since until the time of our operation the other day. He has been in the hospital before, in 1899, when he was suffering from symptoms of stone in the bladder; the bladder was sounded, but without any result. It was therefore thought that his was one of those deceptive cases which are sometimes met with in

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children, and that really there was no stone. On the present occasion, the symptoms of occasional sudden stoppage of the stream of water, pain at the tip of his penis, and the circumstance of his having passed blood on one occasion, as I have mentioned, made the case appear more strongly still to be one of stone in the bladder. We therefore sounded him very carefully, but I could find nothing whatever in the way of objective evidence suggestive of stone in the bladder. It would therefore have been easy to conclude that the case was in all probability one of those deceptive instances of symptoms of stone, leaving no other alternative than to send the boy out and wait upon circumstances, as was done before. But having had a certain amount of experience in cases of this kind, I knew that it did not follow because there was no

FIG. 1.—Showing stone situated in right ureter at level of brim of pelvis. (Mr. Addyman.)

stone in the bladder that these symptoms were not due to stone; in other words, I knew that the same symptoms might be produced by stone not in the bladder, but in the ureter. Having that idea in my mind, we had the boy skiagraphed, and there was found very distinctly, as you will see in the skiagram (Fig. 1), about the level of the brim of the pelvis, a round stone the size of a hazel-nut. Supposing the skiagram had been taken before we had sounded the boy, it might have led to the belief that there was a stone in the bladder, especially if considered in conjunction with the symptoms from which the boy suffered. As it was, we had sounded the boy most carefully, and I knew there was no stone in the bladder; and yet the skiagram showed at the level of the brim of the pelvis a stone as clearly as possible. The stone, in fact, was in the ureter in that situation. The first point, therefore, which I want you to realise is that a stone in the ureter, *provided it is not situated higher up in the tube than the brim of the pelvis*, may produce exactly the symptoms of stone in the bladder; and in old times, before the assistance of X rays was available, no doubt a large number of cases presenting symptoms of stone were set aside as being unfit for treatment, because no stone could be found upon sounding, when in reality a stone did exist, not in the bladder, but in the lower segment of the ureter —an interesting and important matter. In this boy's case, as we were not quite sure, for certain reasons, about the localisation of the stone by the X rays, I thought it wise, first of all on that account, and secondly in order that I might see whether it was possible to extract this stone through the ureter from the bladder, to perform a supra-pubic cystotomy and examine the bladder with these objects. But I found in this case, as I expected before I operated, that the stone was too highly situated to be approached from the inside. The bladder wound was therefore sewn up, and the boy has been set aside until he has healed, when we shall attack the stone from the side, removing it from behind the peritoneum—the best procedure, I think, for dealing with a stone in that locality. So here is a boy nine years of age, who for a long time had suffered from symptoms of stone, the symptoms being due not to stone in the bladder, but to a stone in the ureter, as is very clearly shown by the skiagram.

[The stone was subsequently removed from behind the peritoneum by the combined lumbar-iliac incision; the case was a difficult one in consequence of the way in which the ureter with the impacted stone dropped down into the depths of the pelvis. The stone was situated about 1 inch from the bladder, and was 1 inch long and about $\frac{1}{3}$ inch in diameter (Fig. 2). No attempt was made to suture the ureter, a small drainage-tube being arranged to provide a temporary outlet for the urine escaping from the wound in the ureter. A rapid and perfect recovery followed.]

Not long since I had, outside the hospital, a patient, a man of forty-eight years of age, who for fourteen years had suffered acutely from symptoms of stone in the bladder. Eight years ago he had been cut for stone in the bladder, supra-pubic cystotomy having been performed, and a stone had

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been removed. Later on, as the symptoms were not relieved at all by the first operation, a second operation was performed—that is to say, another supra-pubic cystotomy,—at which it was stated that the mucous membrane of the bladder was found to be covered with phosphatic material, which was scraped away. In spite of these two operations no relief whatever was obtained from the symptoms of stone in the bladder, which consisted of a difficulty in passing water, pain at the end of the penis and a feeling of weight about the perineum, and an occasional sudden stoppage of the stream. These symptoms continued in spite of the two previous operations. I saw him not long ago, eight years after the last operation and fourteen years from the time when, so far as he could recollect, he had first suffered from these symptoms. But upon inquiry it was pretty clear that he had suffered from the symptoms longer than fourteen years. I sounded him most carefully, but there was no stone in the bladder; nothing at all abnormal could be felt. I saw nothing of him again for some months, and in the meantime he had been to several other authorities, one of whom had examined him by the cystoscope and had seen a tumour in the bladder, which it was proposed to remove. Another surgeon had diagnosed atony of the bladder, which he said accounted for the man's symptoms. But nobody seems to have been struck with the idea that a stone in some other position than in the bladder could be the cause of his symptoms. When he returned to me again, seeing that these symptoms continued, I felt confident that somewhere or another, as I could find no other cause for them, there was a stone giving rise to the troubles which he experienced; and bearing in mind that stone in the ureter, if situated below the brim of the pelvis, may give rise to the same symptoms as stone in the bladder in some people, I had a skiagram taken in the same way as we did in the case of the boy. The result of the skiagram was this (Fig. 3). It is a beautiful skiagram, and you can see the stone at the extreme lower end of the right ureter. It was quite clear from my examination that the stone was not in the bladder, and I therefore did a supra-pubic cystotomy and found this large stone quite low down, almost putting its nose into the bladder through the orifice of the ureter on the right side. After a little gentle dilatation of the mouth of the ureter, I was enabled to coax the stone out into the bladder, and so we took it away. The case taken in conjunction with the other one is very instructive, symptoms precisely similar to those produced by stone in the bladder having been produced in each of the cases by calculus impacted low down in the ureter.

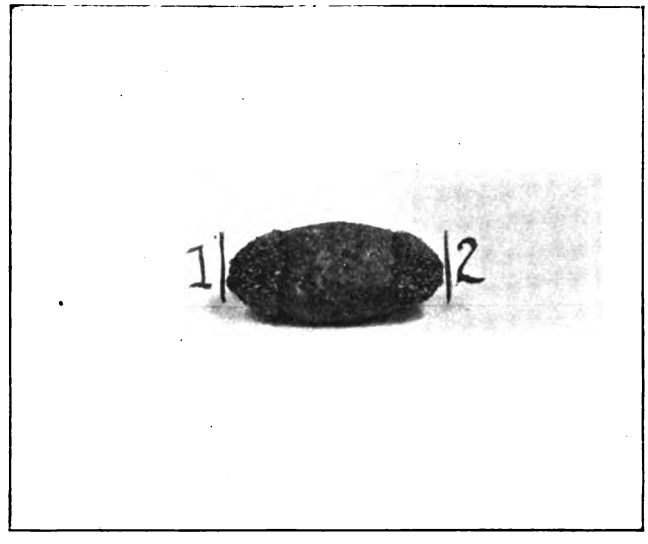

Fig. 2.—Stone shown in Fig. 1 after removal. Natural size.

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Let me relate one other case bearing upon this question. A patient who was, I think, fifty-five years of age, consulted me on account of persistent cystitis, which nothing seemed to cure. He gave a history of having suffered from symptoms of stone for something like twenty-five years. He had been sounded by several people, nobody had detected a stone, and no one thought very seriously of his case, which was taken to be one of those painful instances of chronic cystitis which, although susceptible of some relief by perpetual washing, are sometimes practically incurable, or at least have been thought to be so by some people. Having in my mind the possibility of these symptoms being caused by a stone in the ureter, I had a skiagram taken here also, with the result that a large stone was seen

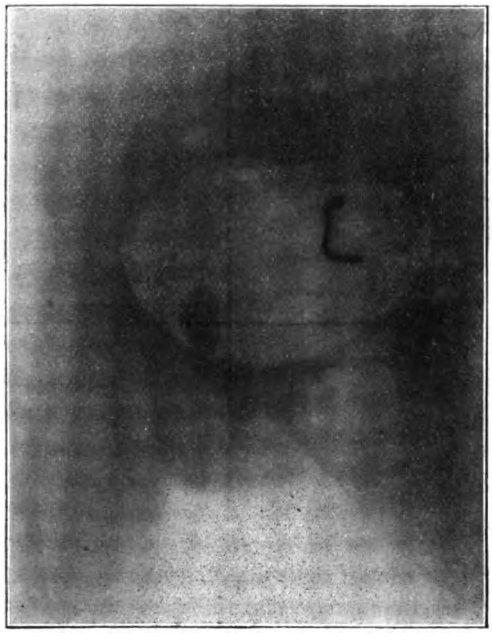

FIG. 3.—Showing stone situated at extreme lower end of right ureter. (Mr. Mackenzie Davidson.)

lying low down in the ureter very close to the bladder, which obviously gave rise to the patient's troubles. In this case, unfortunately, the matter had gone further, the greater portion of the kidney having been destroyed by suppuration, the ureter and pelvis being largely dilated; the stone, in fact, lay in the sac of the large abscess. The dribbling down of the pus and urine by the side of the stone had led to contamination of the bladder, and so produced the persistent cystitis from which the patient suffered. It was obviously useless to attempt to remove the stone through the bladder, because there could be felt in the loin an elongated tumour, clearly composed of the dilated kidney and ureter. I cut down into the loin, opened up the dilated ureter, and found a stone impacted low down about an inch from the bladder. As there seemed to be a considerable amount of the secreting structure of the kidney intact, I stitched the opening in the ureter to the skin wound, hoping that it might ultimately be perhaps possible to save the kidney, as the condition of the urine raised some doubt about the soundness of the opposite organ. Rapid improvement followed in the general health, and the cystitis was soon cured; but as there subsequently seemed little chance of saving the kidney, I removed it with the whole of the ureter, and the patient recovered.

These are three very excellent cases to show the importance of bearing in mind that symptoms of stone in the bladder may be produced by stone situated in the ureter—a fact which must never be absent from your mind when sounding the bladder for stone gives a negative result. This negative result of sounding should always be followed by an X-ray photograph, if the facilities for this are at hand. A bimanual examination should also be made, a finger or two of one hand being in the rectum, and the other hand upon the lower abdominal parietes. In this way it is generally easy in young subjects, and sometimes in adults of moderate stature and not fat, to feel a stone of a size as large as a horse-bean impacted in the ureter at any point below the level of the brim of the true pelvis, the situation in which an impacted stone may give rise to symptoms indistinguishable sometimes from those of stone in the bladder itself. For example, in this boy of whom I have spoken the stone can easily be felt between the finger in the rectum and the hand on the abdominal wall. In the absence of the X rays, therefore, bimanual examination would have been sufficient for a diagnosis in this case. In an adult, of course, supposing the stone is near the brim of the pelvis, it cannot always be reached by the finger in the rectum, but this is sometimes quite possible in thin subjects; and in the case of a stone situated lower down, near the mouth of the ureter, you can feel a stone by bimanual examination in any subject.

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Never omit, therefore, to make a bimanual examination in any of these doubtful cases. I said just now that in one of these cases the result of cystoscopic examination led to the diagnosis of tumour of the bladder; it is quite conceivable that the projection which was caused on the bladder-wall by a stone so low down and so near the orifice of the ureter as the stone was in the case mentioned, might give rise to a deceptive appearance of tumour of the bladder; and no doubt had an operation been proceeded with for the purpose of removing the supposed tumour of the bladder, it would readily have been ascertained that the tumour was not a new growth, but that the deceptive appearance

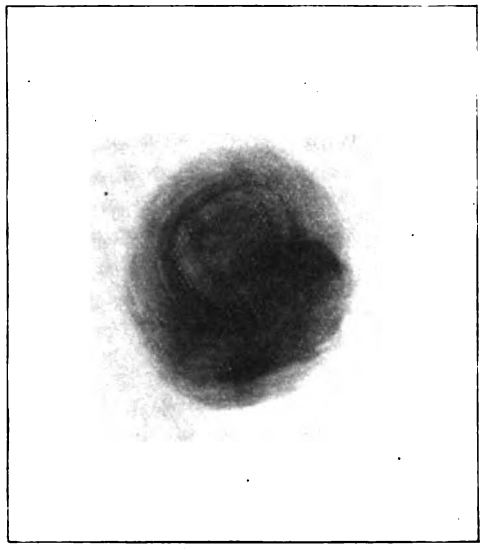

FIG. 4.—Structure of stone in Fig. 3 shown by the X rays. (Mr. Mackenzie Davidson.)

was due to the swelling caused by the stone, so that no great harm need have come of the mistake. From this point of view the case is interesting, as it shows how deceptive the cystoscope may be even in practised hands, and how necessary it is that it should in doubtful cases be supplemented by the use of the X rays. One of the principal symptoms of which these patients complained was pain in the end of the penis, which, as you well know, is one of the commonest and one of the most characteristic pains in connection with tumours, stone, or other foreign bodies in the bladder; and you have, I doubt not, been told that the explanation of this pain is that the bladder as it becomes empty leads to the stone or other mass being forced into the neck of the bladder, thus giving rise to the reflex pain felt at the tip of the penis; indeed, it is sometimes said that unless the stone or other material is capable of being so caught in the neck of the bladder the terminal penile pain cannot occur. If this were true it is clear that by no possibility could a stone in the ureter produce pain at the tip of the penis; but, as a matter of fact, it is not necessary for a stone or any other foreign body to be in actual contact with the neck of the bladder to produce pain at the tip of the penis, because there are other causes entirely outside the bladder which will produce that particular symptom. It is useful to bear in mind this fact, to which I do not recollect having seen any reference, because supposing in a case in which the characteristic pain at the tip of the penis is present you fail by all your methods of examination to elicit evidence of the existence of any stone, other foreign body, or new growth, especially villous growth, in the bladder, you must look further afield for an explanation. That being the case, the question comes, what are the conditions outside the bladder which are liable to give rise to this characteristic pain at the end of the penis which is so liable to simulate stone, etc., in the bladder? This is a matter about which I think you will find very little said in the ordinary books.

The following causes I have met with in my own experience:

(1) Stone impacted low down in the ureter.
(2) Phimosis with single small adhesion.
(3) Fissure of anus.
(4) Rectal polypus.
(5) Sarcoma of inguinal region.
(6) Tuberculous testicle.

Of the first of these causes I have said enough. Simple phimosis with adhesion will sometimes produce this terminal penile pain; and it is an odd thing that the smaller the adhesion the more likely it seems to be to produce this symptom. I once had to do with a case in a young man of twenty-eight, who had suffered for years occasionally, especially when the penis became erect, from the most acute pain at the meatus, so much so—he was married at the time I saw him—that it seriously interfered with the proper carrying out of his conjugal duties. Upon carefully examining the part, by accident I

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discovered a thin string-like adhesion between the dorsal aspect of the glans and the prepuce; and, thinking that this might possibly have been a cause of his trouble, I divided it, and from that day no further pain occurred. This is a good illustration of the way in which a very small thing will sometimes produce a condition not only painful but important, and it further shows how easily the cause of trouble may be overlooked in the absence of an extremely careful examination of all the parts concerned. Fissure of the anus will also sometimes cause precisely the same pain. I have seen three cases in which the predominant symptom in fissure of the anus was pain at the end of the penis, exactly like that produced by stone in the bladder. In point of fact, in two cases the patients consulted surgeons because the probability of the existence of bladder trouble had been suggested, some little discomfort that had been occasionally felt in the rectum when the bowels acted having been overlooked. The cure of the fissure in each case led to the disappearance of the pain at the tip of the penis. The association in children of pain at the end of the penis with rectal polypus is peculiarly deceptive, as it is accompanied by straining and sometimes by prolapse of the bowel; the polypus itself being perhaps too small to attract notice upon superficial examination, the suggestion of vesical stone is very strong. Tuberculous disease of the testis will sometimes produce exactly the same condition. It is difficult to see exactly why this should be, and so far as I know the cases of tubercle of the testicle which are prone to produce pain at the tip of the penis of this type are those in which the body of the testicle is involved; so much so that in any given case of tubercle of the testis which is associated with pain at the tip of the penis, it is a fair inference, so far as my experience goes, that the tubercle involves the body of the testicle itself. I cannot explain to you in the least degree why tubercle of the testicle should give rise to this reflex symptom, but that it does occur in certain cases of this kind I have no doubt. In the two or three cases in which I have removed the testicle for conditions of this sort, the tubercle has not been confined to the epididymis, but has encroached upon the body of the testicle in each case to a very considerable degree; and in one case the tubercle was confined to the body of the testicle. I have not seen the symptom associated with any other disease of the testicle. Returning to the question of stone impacted in the ureter, the question arises as to the proper method of the removal of these stones, for remove them you must. If left *in situ* the occurrence of grave trouble is merely a question of time; the graver trouble being kidney disorganisation with all its evils; the minor trouble being incurable cystitis. Of the more serious result I have already given you an instance in the third case which I have mentioned in this lecture. Of incurable cystitis, the result of calculus impacted at the vesical end of the ureter, I have recently seen several cases. In one the cystitis had existed for nearly twenty years, having resisted every kind of treatment at the hands of many people; there was no evidence of any kind of kidney disease. The cause was finally discovered by the X rays, and I removed the stone, with the result that in about eight weeks from the time of the operation the cystitis had disappeared. With regard to the best method to be pursued in the removal of stones impacted in the ureter, this will depend mainly upon the position of the impacted stone; if situated well above the brim of the true pelvis, the stone is easily removable through the ordinary incision used for lumbar nephrectomy; if lying close to the vesical end of the ureter, it is easily extracted through the bladder after the supra-pubic cystotomy,—its extraction through the vesical orifice of the ureter being rendered more easy by the fact that in such cases the vesical orifice of the ureter is very abnormally patent and lax. When lying below the brim of the true pelvis, and not less than one inch from the vesical orifice, it may be extracted either by a prolongation of the usual nephrectomy incision downwards along the crest of the ilium, or by the sacral route, *i. e.* by an incision passing downwards from the level of the second or third sacral spines to the coccyx (parallel to the spines and three quarters of an inch from them) which gives access to the lower end of the ureter. The former method is much simpler and in all respects more desirable; the transperitoneal method should never be employed; it has nothing to commend it, and there is much to be said against it. This reference to apparently incurable cystitis gives me an excuse for impressing upon you that cystitis is frequently caused by conditions outside the bladder. In any case, therefore, in which the cure of a cystitis is very difficult, never neglect to investigate very

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thoroughly the condition of the kidney, ureter, etc., using the X rays always, with a view to the detection of extra-vesical calculus. Again, please bear in mind that the occurrence and persistence of cystitis is greatly influenced in females by the posture of the patient. In consequence of the flabby condition of the bladder arising in females mainly from the general habit of long retention of urine commonly rendered necessary by social requirements, it is practically impossible for a woman who has reached middle life to empty the bladder completely whilst lying on the back; hence residual urine, generally in large quantities, is almost always present in women confined to bed by illness. Decomposition, rendered more liable in women than in men by the shortness and often half patent condition of the urethra, occurs very readily, giving rise to all the ordinary symptoms of cystitis. Cystitis of this kind, which I have called posture cystitis, is only permanently curable by getting the patient up. In all these cases, as soon as the patient can leave her bed an immediate improvement takes place, and recovery rapidly follows the simplest treatment,—a good practical point to bear in mind, and one which may save you much waste of time and trouble, and the patient great discomfort, if you have a knowledge of it.

Injections of Iodo-ether in Exophthalmic Goitre.—In 'Progressive Medicine' for June, 1903, is found a reference to the paper of Abodie and Collon, on the results reached by them in the treatment of exophthalmic goitre by injections of iodo-ether. The solution employed is 60 grains of iodoform and five ounces of ether. Of this solution, 15 minims is injected in the most prominent part of the tumour, avoiding large vessels. The writers have employed this method in twenty-four cases. Of these, twelve were cured, nine showed permanent improvement, and three a temporary gain. The injections were made with antiseptic precautions, there being usually no subjective disturbances following them, with the exception of sometimes a slight choking sensation or a short dry cough. The ages of the twenty-four patients varied from 15 to 20 years, and the goitres were of all degrees of severity. The injections were given at intervals of a few days, and were commonly followed by prompt improvement in the headache, polyuria, and neuralgic pains. The symptoms more particularly associated with exophthalmos, such as rapid heart and trembling, were markedly relieved.—*Medicine*, September, 1903.

WITH MR. TUBBY IN THE WARDS OF WESTMINSTER HOSPITAL.

OCTOBER 7th, 1902.

SOME CASES OF SURGICAL TUBERCULOSIS AND APPENDICITIS.

GENTLEMEN,—To-day I show you a case which illustrates the subject of surgical tuberculosis, and this very interesting case shows the inception of the disease and the progress it makes.

This patient is a boy aged 20, both of whose parents died young, presumably of phthisis. Sixteen months ago the disease commenced with a swelling situated, as you see, in the first phalanx of the middle finger. This was scraped, and apparently did well, in so far as that it nearly healed. You will notice that the character of the swelling is quite typical. The first point about it is its situation. It is situated in the first phalanx. Two diseases which affect the first phalanx in this way are congenital syphilitic disease and tubercle. These practically are the only two diseases which in young life cause that distinct uniform swelling of the first phalanx. The characteristics of this swelling are well marked. It embraces the whole of the first phalanx, and it seems as though the disease had extended and involved the base of the second phalanx. The swelling is slow, it is chronic and not very painful, and if you feel it you will find that the bone is distinctly swollen.

In earlier cases the bone seems as if it were covered by a thickened periosteum, but in later cases it becomes osteomyelitic, due to the development of tubercular disease in the centre of the bone and gradual extension of the malady to the external surface. This condition, arising either from tubercle or syphilis or osteomyelitis, is known by the quaint old name of "spina ventosa." You will sometimes come across that expression in books. It simply means an expansion of the crust or shell of the compact bone around the cancellous tissue. We come now to the further progress of the disease. It has progressed and is extending towards the second phalanx, causing a certain amount of looseness of the joint. I would also direct your attention to the aspect of that granulating surface. It is prominent and bleeds very readily, and presents all the symptoms or

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appearances of tubercular disease. Leading down from the granulating surface, a sinus goes right into the centre of the bone. Another outbreak was situated on the fourth and fifth metacarpal bones, and the scars which it has left are also typical of tubercular disease. The next outbreak was situated on the dorsal surface of the right forearm. That has been duly scraped and has healed. These outbreaks may not be considered very serious by the ordinary observer, but anyone knowing the history of these cases as they appear in hospitals knows that such outbreaks are the beginning of extremely severe and deadly forms of tuberculosis. The disease is now assuming its more severe form and has broken out in the elbow, forming the typical tubercular elbow with loss of movement, and the usual bulbous outline which is due to several causes. There is much swelling and enlargement of the bone; then there is enlargement and thickening of the synovial membrane and of the subcutaneous tissues, and finally there is wasting of the muscles above and below. This case is a typical tubercular elbow. Unfortunately the disease did not stop there, but there is no doubt that the patient infected himself in the neighbourhood of the inner canthi. He came in with two symmetrical swellings, one on each inner canthus, and it would seem as if he had rubbed his eyes with his fingers and thereby infected the skin there.

The first question, which is of great interest, is as to the primary lesion in this case. There is a distinct history of tubercle in the family, but there is nobody nowadays who believes that tuberculosis and tubercle bacilli are transmitted at birth. Of course, that is different from the question of syphilis, I admit, but I do not think there is any evidence to show that children are born with tubercle actually in them. All we can say on this point is that there is weakness of tissue, which falls an easy prey to the tubercle bacillus. More particularly, from my observation at the Evelina Hospital for Children, I have tried to trace out as far as possible the ways in which tubercle gains access to children. I began by examining the tonsils and adenoids that I took out, and I found in only about three to four per cent. tubercle bacilli. The bacilli, situated in the tonsils and adenoids, are easily carried along the lymphatics and deep cervical glands. Another very important mode of entry for tubercle is undoubtedly the mucous membrane of the nose. Cases which are regarded as chronic rhinitis in children are nothing else, I believe, than cases of incipient tuberculosis of the nasal mucous membrane. The next point is one of great importance. Examinations were made by dental surgeons of standing—I cannot quote the names immediately—of carious teeth removed from children, and in a very large proportion of those carious teeth there were found in the diseased pulp tubercle bacilli. Further examination showed bacilli also in periodontoid tissues and the lymphatic glands. You ask me how it is that the turbercle bacillus gets into mouths so readily. Any one acquainted with the habits of children knows that they will often be found sucking pencils and other things picked up from dirty floors, and thereby tubercle gains an entrance to the body.

There is one particular form of surgical tuberculosis which generally runs a very virulent course, and this is what is known as tubercular dactylitis. Severe cases of surgical tuberculosis frequently begin with tubercular dactylitis. In a large number of cases the infection is by means of a slight wound or injury to the fingers. This lad was a tailor, accustomed to mend old garments as well as make new ones, and I have no doubt that he infected his finger, in the first place, with a foul needle. As showing the severe nature of this form of external tubercle with dactylitis, we have this case in which there are a large number of lesions, and the boy with his tubercular fingers has infected both eyes near the inner canthi. There was a case which came before my notice some years ago of tubercular dactylitis which commenced in the middle finger in the same way; it was followed by tubercular lymphangitis of the anterior aspect of the forearm, and this was succeeded by a tubercular elbow, tubercular glands of the axilla, tubercular pleurisy, and phthisis. I quote that because it shows the extremely severe nature of the disease. So serious did I consider it, that when I found it was in the axilla I proposed to remove the upper extremity. Subsequently the case was operated upon a large number of times, but the patient ultimately died of tubercular meningitis.

I need not go into the question of prognosis. This will be formed upon the history and situation of the lesion, the non-responsiveness of the patient to treatment, and the tendency of the

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disease to spread continuously. Tubercular infection spreads in the same way as a septic lesion, and especially through the lymphatics.

We must come more particularly to the question of treatment, and here we are faced by one very great trouble. I believe I am expressing a certain amount of truth when I say that the treatment of tuberculosis depends upon the patient's surroundings, his social position, and the help he can command. I should say, speaking broadly, that no one of those cases should be treated in any urban hospital. I think there is a very large waste of time and money if these cases are taken into such hospitals. I should like each hospital in London to have its own country establishment, where these patients could be sent. The operations might be done in London, and then the patient drafted to one of these places away from the city. As an illustration I might quote a case under my care in private for ten years. She has had fresh outbreaks of the disease at different times. The disease started with tubercular dactylitis in the left middle finger in the first phalanx. We scraped that out and sent her to Ramsgate, and she became well for a time. The next outbreak was tuberculosis of the right humerus. That got well after a considerable time. The next lesion was spinal caries, which came on quite insidiously. First of all she complained of pain in the back and down the leg. I soon found that she had spinal caries and a large abscess. I opened the abscess and treated it after the most careful aseptic methods. It healed after about three months, and apparently is perfectly well. We come to another outbreak when she had tubercular eruption round the nose. That I cauterised, and it did well. She developed last year two small tubercular places on the skin of the right thigh ; these I excised quite freely so as to get rid of the trouble. I saw her the other day, and she has another slight outbreak of tubercle. She is now a woman of twenty, and looks extremely well. You would say she looked the picture of health, being quite ruddy. But this case is an example of what may be done by surgical treatment when the patient has social means and suitable surroundings. My contention is this —and I hope to make it good by a series of statistics which I have been collecting, — that patients suffering from this form of tuberculosis ought to be sent away for treatment to the seaside or to the country. This holds out the only hope that we have for effectual treatment. It is perfectly useless to keep them here in London. We are compelled to keep this boy because no convalescent home will take him, as he has sores about him which must be dressed. That is a pitiful state of affairs.

The next case I show you is another example of what may be called surgical tuberculosis, the term embracing diseases of the skin, the mucous membrane, the bones, and joints. This is a case of hip-joint disease in what is known as the second stage, namely, the stage of disorganisation of the joint with fixation, loss of flexion power and adduction. She has been in the hospital for some time, and you will find on examining her that the diseased part is, first of all, very tender ; secondly, you can feel a distinct fulness over the head of the femur ; thirdly, there is fulness in Scarpa's triangle. The inevitable outcome if she remains in London will be that she will get into the condition of the case in Luke Ward. The interesting point about the case is that she came in with an extreme amount of deformity. The deformity in many cases of hip-joint disease is not fixed. It is largely due to muscular spasm. This patient was placed in bed and extension was gradually applied. The result is that to-day much of the deformity is seen to have disappeared. With regard to the application of extension, there is one very interesting point that I want to bring out, and which was first put forward by Mr. Howard Marsh. He showed that if a child comes in with the flexed or abducted hip-joint, it is no good to attempt to apply extension with the limb in its apparent position. You must put the hip-joint and limb into the true position, and you should do so in this way. You first of all place the pelvis so that the two anterior superior spines are at right angles to the mid-line of the body. Then place the sound leg parallel with the median line of the body. In the next place you make sure that the lumbar spines are flat on the bed, and this is done by flexing at the hip until the spine is flat. The point I wish to enforce is that if you wish to get rid of the spasm very quickly you must apply your extension along the true, not the apparent line. Let us take the result of applying it along the apparent line. What you do is to place the leg first of all in a false position and lose

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all idea of what is its real position. In the next place, if you attempt to apply extension along the false line of the leg you have a lumbar lordosis and tension of the psoas muscle, and the force you apply simply results in pushing the head of the femur more and more firmly into the acetabulum. My contention is that, in order to get sufficient relaxation of the psoas muscle, you must put the leg into the true position and then apply the force. If you do this you will find most hip-joints come down straight to the bed and parallel with the other leg in the course of a fortnight or a month. That is a very important point to know. Shortly, what I mean is this,—lift the leg until the spine is flat on the bed, and until the two anterior superior spines, with the line adjoining them, are at right angles to the mid-line of the body, and then you get a good result. Day by day relax the position more and more until the leg comes flat down on the bed.

This patient, Lizzie T—, æt. 6½ years, was readmitted to Westminster Hospital on August 15th, 1902. She was an in-patient under my care from October 1st, 1901, to November 13th, 1901, with coxitis of the left side. Her condition improved with rest, and she was sent out with the Thomas's hip splint. The family history was markedly tubercular. On admission there was evidence of very extensive disorganisation of the hip-joint, and there was a large abscess situated on the anterior aspect of the hip. The child's condition was bad, and so it was deemed advisable to open the abscess only. This was done on August 18th, and the condition of the parts was found to be very bad. The head of the femur was completely eroded, and the disease had extended down into the shaft of the bone, so as to give rise to the condition of tubercular osteomyelitis. The pelvis was also extensively involved, and the ilium was greatly thickened. As the disease continued to progress, amputation of the hip-bone was proposed to the parents, but it was refused. I am anxious that you should see this child, because this is another instance of surgical tuberculosis, and unfortunately it is a very bad instance. In connection with these cases there is one interesting point. In hip disease where you suspect pus it is important to examine the cases *per rectum*, because the key to the existence of the presence of pelvic abscess is generally found in such an examination, and it is an important point in the prognosis of hip-joint disease. By examining the rectum you can ascertain how far the disease has affected the pelvis. The question of the infection of the pelvis with hip-joint disease is a most important point—probably next in importance to the question of the infection of the shaft of the femur by osteolitis.

This is a case of a different character altogether. The child is aged eleven years. She was sent to the hospital to be operated upon for appendicitis. The history is that in the beginning of August the patient complained of pain in the stomach and sickness. Her stomach was swollen, and she had first diarrhœa, and then became constipated. The pains were intermittent. She was advised to come to the hospital. The previous history does not throw very much light on the case. When young she had inflammation of the lungs and measles. She seems to have had similar attacks when four years of age, with pain in the stomach and sickness, she brought everything back, and the smell of it was said to be very offensive. She had an attack of pain in the chest two years later. In her eighth year she suffered again from pain in the stomach, so that she appears to have had several attacks of pain, probably due to a diseased appendix. From this you will probably infer that the disease which she suffered from was of an acute nature, and your thoughts naturally run to appendicitis. The question is, are we justified in operating for relapsing appendicitis? I have not seen the child in an acute attack. I have not myself seen any lump nor swelling there; but, on the other hand, there is a distinct history of the trouble. I wish I could feel, as you can in some cases of appendicitis, a distinctly thickened appendix. The way in which you should feel for it is this:—You place the patient on the back, raise the legs, tell the patient to take a deep breath and open his mouth. You then insinuate the tips of your fingers, and press backwards and a little downwards. In cases in which the appendix is thickened you will feel it as a distinct definite cord crossing over the psoas muscle. In this child I cannot feel it. There are two fallacies about feeling the appendix. You may mistake the psoas minor muscle or the ureter for it; but if you feel a definitely thickened cord on deep palpation, and if you have a history of previous

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attacks, you are probably dealing with a thickened appendix. That is quite a sufficient justification for operating on relapsing appendicitis. The reason you cannot find the appendix in children is that in children the appendix very commonly hangs down the pelvis, and can only be felt *per rectum*, and not by abdominal palpation; as it hangs down in children pelvic abscesses are frequent. That is not a bad thing altogether, because a certain number of these abscesses open spontaneously into the rectum. What is the condition of the appendix in a child who has had a number of serious attacks of appendicitis? It is interesting to know the pathology, because it guides you as to the urgency or otherwise of operation. In a child who has had one slight attack of appendicitis you constantly find the appendix thickened at one spot. Its canal is narrowed at that particular spot, and the appendix above is somewhat distended and dilated. In a patient who has had more than one attack you will see the appendix thickened and irregular, and sometimes you will find a distinct cystic swelling at the further extremity of it. If you open the cystic swelling there will come out of it some foul-smelling fluid, though often quite clear. That fluid gives a pure culture of the *Bacillus coli*. In other cases you will find a different condition. I have seen such in a girl aged fourteen, who had had seven distinct attacks of appendicitis. Her appendix was very much thickened and I removed it. When I cut it open I found a small ulcer a quarter of an inch across, and the base of it was so thin, being formed of peritoneum and nothing else, that I take it, if the patient had had another attack, there would have been perforation and an end to the case. So that practically the condition you meet with in these recurrent cases of appendicitis is a thickened appendix, or a constricted or twisted appendix, or a cystic condition or ulceration of the appendix.

There is no disease which possesses so much interest as appendicitis. You may get an acute and most virulent form, a fatal form, from a diseased appendix in which there is no perforation whatever. In some cases you find that the submucous tissue of the appendix is swarming with bacilli coli and other micro-organisms, which may pass from it into the peritoneal cavity. You will find more often that, though in the appendix there is no breach of the peritoneum, there is a submucous abscess, and infection is carried from that. So that the amount of peritoneal inflammation from appendicitis cannot in any way be measured by the condition of the appendix, only by the potential virulence of the *Bacillus coli*. Other bacilli sometimes found in the appendix are the pneumococcus and the *Staphylococcus pyogenes*. But in 95 to 97 per cent. of the cases you get the *Bacillus coli communis*.

A few points of practical interest with regard to operation may be spoken of. In former days, when an abdomen was opened for appendicitis, the the usual thing was to make an oblique incision and cut straight down to the peritoneum. But it began to be found that a large number of cases operated upon for appendicitis showed either weakness of the abdominal wall or very bad forms of ventral hernia afterwards. As a result of operation, I have seen some ventral herniæ where half the abdominal contents have been outside. That is a most difficult form of hernia to treat. It is not really capable of being treated by radical operation, because around the orifice of the hernia the tissues are all stretched, and the sutures will not hold. Statistics show that in 85 per cent. of the cases operated upon in the old way there was either distinct bulging of the abdominal walls or ventral hernia. The only drawback in the method of operation that was devised by McBurney is that it takes twenty minutes longer to do it. The first incision is made through the skin obliquely with its centre crossing McBurney's point. I prefer to go a little bit outside that. The incision passes through the skin and the external oblique, which is thus divided parallel to its fibres. The fibres of the internal oblique and transversalis are then split, and finally the peritoneum, which you open along the line of the external incision. The drawback is this: the skin incision has to be large; and the space for getting out the appendix is limited. But you get over these difficulties with practice. The appendix is removed, and then each layer is sewn up separately. The result is that you have a perfect scar, and you do not get the after trouble of bulging and hernia. With regard to the position of the appendix, in children it often drops down to the pelvis, and should be sought for there. In other cases, more commonly in adults, the appendix points towards the umbilicus. In some

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cases it is very long, and trends towards the left side. I should like to mention that you may have a distinct left-sided appendicitis. In July I saw a lady who had a history of appendicitis. She had a swelling on the left side in the middle line situated above the iliac fossa. The diagnosis was not very clear because of the position of the swelling. I said it was one of two things. She had a distinctly tubercular history. Either it was tuberculosis or appendicitis. We opened the abscess, and with my finger I could feel the appendix passing from the right side as a distinct cord and entering the abscess cavity. I have seen appendicular abscesses under the liver, and in the loins, and behind the cæcum and colon. So that you must recognise, in operating for a case of relapsing appendicitis, that you may find the appendix in any of these positions, and it may be bound down, and the operation may vary from the easiest to one of the most difficult. A child who had had very bad appendicitis on three occasions was brought to me for operation. I had no reason to believe that it was going to be difficult. I came down on the peritoneum and found the peritoneum and cæcum adherent, and there were adhesions all round. I could feel the appendix behind the cæcum, but found it absolutely impossible to reach the appendix without tearing the cæcum. Finally I gave up the case because of the danger. The greater the number of attacks, the more adhesions, and the greater the difficulty of operation. If you operate too soon after an acute attack, when you come to the cæcum and appendix you will often find them so soft that they are very difficult to handle without rupture. I think, therefore, that the earliest period you ought to operate after an attack is three weeks. You must take great care in the interval to keep the patient quiet.

Mental Aberration Consequent Upon Pelvic Disease.—L. G. Hanley ('N. Y. Med. Journ.') reports ten cases of mental aberration cured by operations upon the pelvic organs. Six of the cases had dysmenorrhœa, four had diseased ovaries. In three cases endometritis was present. He believes that no woman should be committed to an insane asylum without first receiving a thorough examination of her pelvic organs and having treated any trouble found.—*American Journal of Obstetrics*, September, 1903.

THE DIFFERENTIAL DIAGNOSIS AND TREATMENT OF CHRONIC DISEASES OF THE JOINTS.*

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Lecture II.

Treatment of Chronic Gouty Affections of the Joints.

Quite apart from the treatment of an attack of gout, which is a comparatively simple and easy matter, must be considered the treatment of the condition or conditions which led up to the attack. In connection with this point, it must be remembered that the gouty individual is one whose general metabolism is unstable, and that this instability may be present in one or more of the great physiological systems—the digestive, the nervous, the circulatory, etc. It should always be a matter for patient investigation as to which of these systems is primarily and mainly at fault, and then to beneficially influence the metabolism of that system by suitable medicinal, dietetic, and hygienic treatment.

In addition to colchicum, which may be given in small doses, guaiacum may very usefully be administered as an alterative which stimulates the metabolism of the liver, and also affords relief to the portal system. From five to ten grains of guaiacum resin should be given in cachets two or three times a day. If constipation occur, a sulphur and guaiacum tablet, or a dose of compound liquorice powder, should be taken at night. The elimination of uric acid may be promoted by encouraging free diuresis by the drinking of sufficient quantities of water, and by the administration of citrate of potassium. The use of the citrate of potassium may with advantage be pushed until moderate alkalinity of the urine is produced.

The use of alkalies and the salts of alkalies.—Of the potassium salts used in the treatment of gout, the citrate and the bicarbonate are the two most commonly employed. As to the beneficial effects of employing a potassium salt in conjunc-

* Two lectures delivered at the Medical Graduate's College and Polyclinic on May 8th and 12th, 1903.

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tion with colchicum, in the treatment of acute and subacute gout, I am fully assured, and my experience is, that of the various potassium salts the citrate is the most useful. If given in sufficiently large doses, it tends, by its conversion in the kidneys into the carbonate, to diminish the acidity of the urine, which is generally high in connection with the gouty paroxysm, while at the same time it increases the solvent power of the urine for the uric acid salts, and so assists their elimination. In addition, as I have experimentally shown, the presence of a potassium salt both delays and inhibits the conversion of the soluble gelatinous sodium biurate, which is the form in which the biurate is first present in the blood, into the comparatively insoluble crystalline biurate. In this way the deposition of the latter in the tissues is inhibited, and so further time for the elimination of the biurate is offered. I do not, however, wish to contend that the above-mentioned are the only explanations of the beneficial action of potassium salts in the treatment of gout. In addition they have their uses in the gouty state, on account of their stimulating action upon metabolism, of their remedial action upon the gastric and hepatic functions, and of their diuretic action.

As regards the use of lithium salts in the treatment of gout, my opinion is that they are not so useful as the potassium and sodium salts. The lithium salts have not the same inhibiting effect on the conversion of the gelatinous sodium biurate into the crystalline form as the potassium salts have, while at the same time they have no better solvent effect on gouty deposits. The great objections, however, to the use of the lithium salts is their greater toxicity and depressing action on the heart, as compared with the potassium salts. They consequently have to be given in such small doses that I am very doubtful as to whether in such doses they possess any remedial effect at all. On the other hand, I constantly meet with patients suffering from cardiac depression as the result of the excessive and continued consumption of lithia tablets, which are so persistently, so speciously, and so wrongly vaunted as curative of gout.

In the treatment of the gouty state associated with disturbance of the functions of the liver, the most important consideration is the restoration of that organ to its normal state of activity, and here the alkaline sodium salts are especially useful. There is no better treatment at the outset than a dose of blue pill or calomel at night, followed by a dose of Epsom salts or Carlsbad salts in the morning. Subsequently, a pill containing a small dose of blue pill or calomel, combined with euonymin and colocynth, will be found most useful. In such cases of gouty hepatic inadequacy a mixture which I have found most beneficial as regards its stimulating effect on the metabolism of the liver, and also of the gastro-intestinal tract, is one containing sodium bicarbonate, gentian, and nux vomica, taken a quarter of an hour before meals.

Baths and massage.—A sedentary life and deficient exercise conduce to gout, owing to a lowering of general metabolism, to depressed vigour of blood circulation, and to impairment of movement of the lymph in the lymph channels. Since the lymph circulation is mainly carried on by muscular movements and respiration, the beneficial influence of massage and muscular movements on many gouty subjects becomes readily intelligible. Massage should never be resorted to in cases of acute gout, as such treatment not only aggravates the disease at that stage, but also causes severe pain; it should be reserved for the more chronic cases. Massage produces an increase in the amount of blood and lymph passing through the tissues concerned, both during massage and after its cessation. This increased circulation improves the nutrition of the affected tissues, promotes absorption of deposits, and restores physiological activity. In cases of subacute or chronic gout, where the joints remain swollen and œdematous, and are the seat of considerable deposits, much benefit is frequently derived from massage and galvanism. The affected joints should be massaged, each one for a few minutes, and then galvanism (five to ten milliampères) applied to each joint for a few minutes, with the negative pole over the affected region. The galvanism is then to be followed by a few minutes' massage. Under this combined treatment the œdema and the deposits frequently disappear rapidly. Probably the beneficial effects are due mainly to the increased circulation of blood and lymph induced, and the consequent absorption that takes place. I have also found in many cases that a decidedly beneficial influence on gouty joints is produced by radiant heat baths,

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followed by electrical treatment in the form of cataphoresis to the affected joints.

Radiant heat and superheated air baths increase the oxidative processes within the body, as is shown by the increased elimination of carbon dioxide from the lungs, and also by the increased metabolism of the body in general. They also stimulate the circulation both of the blood and lymph in the affected joints, and so lead to improved nutrition of the parts. This curative action undoubtedly continues after the treatment has been left off. Therefore such treatment is better intermittent in its application—say six baths on alternate days, and then interrupted for two or three weeks, and so on. These baths also improve the atrophy of the muscles. They cause a temporary elevation of the body temperature, marked reddening of the skin of the part treated, profuse local or even general respiration, quickened pulse, lowered arterial tension, and generally considerable amelioration of the pain, and in some cases complete disappearance of it for a time. Radiant heat has a greater penetrative effect than other forms of heat, and in my opinion the effect is more stimulant. In cases of acute or subacute gout, the pain, as a rule, recurs at varying intervals after a bath, but usually with diminished severity, and in favourable cases a progressive reduction of the pain occurs after the use of subsequent baths. Undoubtedly many cases of chronic gout do not show much improvement after the use of radiant heat or superheated air baths, and I have frequently experienced great difficulty in selecting the cases most likely to be benefited by it. As a general rule my experience has been that cases of chronic gout of long standing, with considerable deposits in the joints, do not derive much benefit from these baths. For such cases undoubtedly much more good can be done by the employment of vapour baths, followed by massage of the affected joints, a method of treatment which is frequently most useful in producing softening and absorption of the deposits. It should, however, be borne in mind that radiant heat baths seem to set up improved circulatory and trophic changes in the joints, which apparently are maintained for a prolonged period of time after the baths have been discontinued. Certainly I have seen in several cases, after a course of twelve or eighteen radiant heat baths had resulted in but slight improvement, and the baths had been abandoned in despair, a progressive improvement maintained for weeks and even months after the discontinuance of the baths, an improvement which in some cases has progressed to more or less complete cure. If only one limb or a part of a limb is the seat of gout, the question arises as to whether that limb should be locally treated by being placed in a small specially constructed bath, or whether the entire body bath should be used. My experience is that the whole body bath is in all cases the more useful, with, in the case of the radiant heat bath, an extra localisation of the heat and light rays on the affected part. That means, according to my experience, the more extensive the surface to which the heat and light rays are applied the better is the result.

When the ordinary radiant heat or superheated air baths are not obtainable, very good results may be obtained at home by the use of an ordinary blanket tent with a small opening at the top to let out the hot air saturated with moisture. The hot air is supplied by a ring Bunsen gas burner, or by a large spirit lamp with a flue passing through an opening in a blanket at the foot of the tent.

Cataphoresis is useful in many cases of chronic gout with considerable deposits in the joints, and also in many cases of obstinate rheumatoid arthritis. It consists of the electric transfer of fluids through the joints from anode to cathode. The best fluid to use in cases of gout is a solution of potassium bicarbonate. The padded negative electrode should be placed on the back or chest, and the positive electrode, which should be button-shaped and padded, and which is kept wet with the saturated solution, should be applied to the joint. Currents of from 10 to 30 milliampères may be employed.

Treatment of Rheumatoid Arthritis.

Rheumatoid arthritis, if left untreated, tends to spread from joint to joint, and produces progressive destruction of the joint tissues. Occasionally treatment fails to effect any arrest of the disease, and this is especially apt to occur in connection with the rheumatoid arthritis of the old. For the successful treatment of this disease it is essential that the treatment should be commenced while the disease is in its early stages; hence the importance of an early recognition of the malady, and of its distinction from gout and rheumatism. The treat-

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ment must be persevered in for a lengthened period of time, generally a year or two, and during the treatment everything possible must be done to increase the patient's strength.

If rheumatoid arthritis is seen and recognised early in the acute stage it is curable. In the latter chronic stages it is possible to arrest the disease, to remove the pain, and to secure greater movement of the joints, but it is not possible to bring the disorganised and deformed joints back to their normal state. The not infrequent mistake of diagnosing rheumatoid arthritis as gout, and the consequent placing of the patient on a restricted and spare diet, has undoubtedly led to the development of severe and incurable forms of the disease. The diet should be as liberal and as good as the patient can digest, and animal food should be partaken of freely, though not to the exclusion of vegetables. A moderate quantity of wine or stout should be taken with lunch and dinner. It is essentially a disease that requires good and nutritious feeding, and I have seen many cases of rheumatoid arthritis which have gone thoroughly to the bad, through the initial error of mistaking the disease for gout, and treating it with a spare diet. Woollen clothing should always be worn next the skin, and exercise, short of producing pain, should be indulged in. A dry gravel soil and a warm dry climate are most suitable to patients suffering from this disease.

The treatment of rheumatoid arthritis by drugs must be quite different from that of gout or rheumatism, and efficient measures must be taken to improve the general condition and health of the patient. The drugs that I have found most useful in the treatment of rheumatoid arthritis are guaiacol and potassium iodide. I have now employed guaiacol in a very large number of cases extending over several years, and as the result of my experience I do not hesitate to say that, if administered in sufficient quantities and for a sufficiently long period of time, it is capable in the great majority of cases of arresting the disease, of diminishing the size of the joints, and of permitting increased movements. It also relieves pain markedly. It is useful in both the acute and chronic forms of rheumatoid arthritis. I know of no satisfactory explanation of its action, but it probably acts on the intestinal contents by arresting abnormal fermentation, and on the bacterial toxic products. The most convenient form of administering it is the carbonate of guaiacol in cachets. This salt is a white powder which is free from the disagreeable odour, taste, and irritating effects on the stomach of guaiacol itself. In the intestines it is slowly split up into guaiacol and carbonic acid gas. At first five grains of the carbonate of guaiacol should be given three times a day, and the dose should be gradually increased until from fifteen to twenty grains are being taken in each dose. It is essential that this treatment should be continued for at least twelve months. The beneficial effects of the guaiacol are very much increased by administering at the same time a mixture containing moderate doses of potassium iodide; the depressing effect of the iodide should be counteracted by its combination with tonics, of which perhaps nux vomica and the compound glycerophosphate syrup are the most useful. In my experience arsenic is of doubtful value in the treatment of this disease. If the joints are very painful the application of salicylate of methyl, or painting with iodine, followed by the application of hot linseed poultices, or blistering in the vicinity of the joints, are all useful measures.

The thermal treatment of the affected joints, either by means of baths, superheated air, or radiant heat, is most beneficial. Douche massage is the most effective form of treatment with hot water, and perhaps next to that ranks the peat bath, such as can be obtained at Strathpeffer. Radiant heat baths, in which the affected joints are bathed in the heat and light rays reflected from a number of incandescent electric lamps, are also beneficial in many cases. Electric baths, using the alternating current either from an alternating dynamo supply or from an induction coil, are also most useful in the early stages of rheumatoid arthritis.

Properly regulated movements and properly applied massage are of great use in overcoming the stiffening and fixation of the joints, and the muscular wasting in their vicinity. Massage, in addition to its local influence upon the affected joints and their proximate muscles, also improves the general circulation and the general nutrition of the body. General massage should therefore be lightly applied at first, little or no attention being paid to massage of the affected joints for the first few days. The form of manipulation which may be applied to the joints with the best results is massage à friction, which consists of the application of quick frictions

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or rubbings to the surfaces of the joints. In addition, gentle kneading and squeezing of the parts, particularly of the tendons and fibrous surroundings, should be effected. The effects of such manipulations are generally evidenced by the rapid absorption of exudative products in and around the joints. Active and passive movements of the affected joints should also be employed.

If possible, a patient suffering from rheumatoid arthritis should not winter in this country. A dry warm atmosphere is required, which can be best obtained in Egypt and Algeria.

If the locking of a joint is extreme, and is otherwise incurable, it may be advisable to excise the joint. Successful excisions of the elbow and knee in cases of rheumatoid arthritis have been performed.

Treatment of the other Forms of Chronic Joint Disease.

Senile arthropathies.—These are best treated by baths, massage, the administration of potassium iodide, and in suitable cases by occasional blistering in the vicinity of the affected joint.

Chronic joint disease in children, described by Still.—In this disease the only treatment that appears to be of use is careful attention to diet and hygiene. Recovery of the joints may occur.

Gonorrhœal arthritis.—The point of origin of the general infection should, if possible, be ascertained,—in the urethra from the meatus to the prostate, the glands of Littré, the vesiculæ, Bartholin's gland, and the uterus in women. Suitable treatment should then be employed. The calibre of the urethra, if diminished by stricture or atresia, must be restored. Urethritis and prostatitis, if present, must be treated.

In addition to local treatment of the urethra and prostate, the administration of urotropin is very beneficial. As regards the local treatment of the joint affection, if there is simply pain without swelling, it is only necessary to ensure rest by fixation of the joint, and to apply a sedative lotion. Later on a local hot air or radiant heat bath may be usefully employed. If the arthritis is very acute, or if there is persistence of much effusion, arthrotomy should be performed freely, followed by antiseptic irrigation and drainage.

Quiet effusion into knee-joints.—In the treatment of this affection splinting and rest of the joint should be avoided. On the contrary, massage should be employed, and exercise in the open air encouraged. Attention should also be paid to the menstrual irregularity or uterine troubles that are generally associated with this affection.

Nerve degenerative arthropathies.—In addition to the treatment of the causative disease of the central nervous system, cupping or blistering along the spine is very useful, and in order to keep up the counter-irritation it is advisable in many cases to keep open the blistered surfaces for some days by dressing with savin ointment.

Tuberculous disease.—The constitutional disease must be suitably treated, and the affected joint or joints rested. The disease of the joints is always very protracted, and although ankylosis may occur, yet, on the other hand, a certain amount of movement may be secured. Resection of the joint results in more speedy recovery, but of course with an ankylosed joint.

Syphilitic joint disease.—The joint symptoms quickly disappear under treatment with large doses of iodide of potassium, combined with mercurial inunction.

Joint changes in Raynaud's disease.—These cases are much aided by balneological treatment, together with the use of sedative drugs to the nervous system combined with small doses of nitroglycerine.

Rheumatism v. Muscular and Joint Pains.—James J. Walsh says that many cases with the diagnosis of rheumatism are examples of muscular and joint pains due to quite other causes. Among the most prominent causes are: flat-foot, occupation brachyalgias, and shoulder pains of various kinds due to the excessive or faulty use of muscles. In neurotic conditions pain is a very relative affair. In highly excited neurotic states even a little discomfort may seem almost unbearable pain. A case of tabes came under the notice of the writer in which the lightning pains took the form of sciatica and were treated as sciatica-rheumatism. A very interesting set of cases of pseudo-rheumatism were noted as occurring in connection with scoliosis. In one case joint symptoms occurred in an arthritic neurosis of the knee, consequent upon the pinching of a loose cartilage in the joint.—*Medical Record*, September 19th, 1903.

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